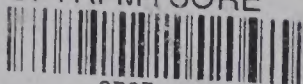


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Nutrition in Ind.





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# NUTRITION IN INDIA









MAP OF  
THE INDIAN REPUBLIC

# NUTRITION IN INDIA

BY

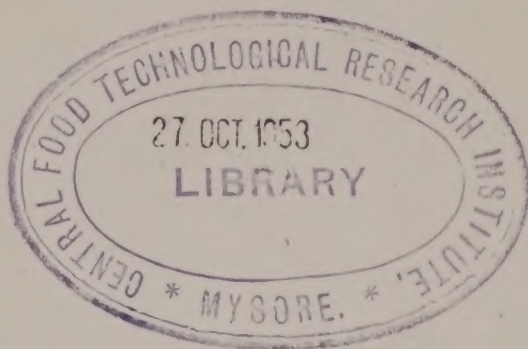
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INDIAN COUNCIL OF MEDICAL RESEARCH

COONOR, SOUTH INDIA



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## P R E F A C E

THIS BOOK is a review of the work done in India in Nutrition and allied subjects during the last forty years or more. The nutritive value of Indian food-stuffs, diets and dietary habits, nutritional disorders and diseases, nutrition in public health practice, food supply and population are some of the principal subjects covered in this book. It is not a text book of nutrition for students. It is mainly intended for research workers in India and abroad. In writing this account, several gaps in our knowledge have been found to exist and attention is drawn to them in the hope that further activity in these fields would be stimulated. At the same time it was felt that a book of this type would indicate the progress made in this country so far as nutrition research and public health nutrition were concerned. The author is aware that several books on food and nutrition in India have been published in the last thirty years, but few of them have taken into account the vast amount of work that has been done in India. Workers abroad must have felt at times the difficulty of getting at the original references on several aspects of nutrition, work on which has been published in India. This book with its extensive bibliography will not only help to bring the Indian work before the world but will also give access to some valuable publications in nutrition.

The author has great pleasure in gratefully acknowledging the generous assistance given by Dr. V. R. Khanolkar, Director, Indian Cancer Research Centre, Bombay, and the Editorial Board of the Indian

Journal of Medical Sciences, Bombay, in sponsoring the publication of this book. He is also grateful to his colleagues Dr. C. Gopalan and Dr. V. Ramalingaswami for the many valuable suggestions made by them. The author's thanks are due to the Editors of the Indian Journal of Medical Research and the Indian Medical Gazette for permitting reproduction of some figures and tables from the respective journals.

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COUNCIL OF MEDICAL  
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*5th September 1952*

V. N. PATWARDHAN

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## CHAPTER I

### INTRODUCTION

THE FIRST DECADE of the twentieth century saw the commencement of nutrition research in India. McCarrison and McCay were the pioneers in the field and till 1920 were the only investigators in India actively interested in nutritional science. Curiously enough both were impressed by the fact that the physique and health of Indians varied in different parts of India. Both of them were convinced that the underlying cause was dietary. McCay devoted his attention to the protein element in nutrition. His careful and painstaking researches (described later) were intended to show that the proteins of inferior quality, both from the standpoint of digestion and utilisation were responsible for poor physique and stamina of certain Indian population groups. McCarrison's concept of the role of diet was more comprehensive. In keeping with the advances in nutritional science made in other countries, McCarrison formed the opinion that in certain Indian diets there were multiple defects, and that these defects were the cause of poor physical development, low resistance to disease and widespread illhealth in India. McCarrison's researches made it quite clear that he was least interested in the deficiencies of single nutrients and their effects. He planned his experiments so as to test the effects of natural dietaries with multiple defects of deficiency and imbalance on the physiological function and structure of body organs, in order that such knowledge may be used to find explanations based on scientific observations, for the state of health

in human beings. It was only towards the end of McCarrison's active career in India that nutrition researches in India swung over to the study of individual nutrients. It is somewhat surprising that although illhealth in India was recognised to have malnutrition as one of its important causes, very little attention was paid to clinical nutrition till well on into the fourth decade. Beri-beri and goitre were of course two main exceptions, for it cannot be denied that McCarrison had devoted a great deal of attention to the study of these two diseases. It is only recently, however, that clinical researches in nutrition have made any headway, this despite the fact that clinical material for such purposes has always been available in plenty.

India owes it to McCarrison that he not only founded a school of nutrition research in this country but by his brilliant researches helped to attract other scientific workers to the field of nutrition research. It would be no exaggeration to say that McCarrison laid the foundation on which the present structure of nutrition research in India has been built up. In this connection mention must be made of the Indian Research Fund Association, a semi-official organisation which came into being in 1911. Among its objects were the two following:

1. The prosecution and assistance of research, the propagation of knowledge, and experimental measures generally, in connection with the causation, mode of spread, and prevention of diseases, primarily those of a communicable nature.
2. To initiate, aid, develop and co-ordinate medical scientific research in India and to promote and assist institutions for the study



of diseases, their prevention, causation and remedy.

The Association derived its funds from the Government of India in the form of annual grants, but was fortunately entitled to spend them in its own discretion for the furtherance of its aims and objects. There is no doubt that the credit for fostering research in medical and allied sciences during the last forty years goes almost entirely to the Indian Research Fund Association. Towards the end of 1949, the Association took on a new and more apt name of Indian Council of Medical Research.

McCarrison's early researches dealt with endemic goitre which he observed in 1904 in Gilgit on the far north-west frontier of undivided India. His interest in goitre was lasting and the investigations on thyroid gland occupied a major portion of his time during his scientific career in India. In 1913-14 McCarrison began his researches at Kasauli (Punjab) on the relationship between diet and disease under the auspices of the Indian Research Fund Association. A few years later, in 1918 to be precise, he moved down south to Coonoor and began his researches on beriberi. Within a few years the scope of his activities widened and his unit at Coonoor became known as 'Deficiency Diseases Inquiry'. The Royal Commission on Agriculture appointed to report on the Indian agricultural practices and policy was keenly interested in McCarrison's work. In 1926, they paid a visit to Coonoor to acquaint themselves with McCarrison's work at first hand. They fully realized that nutrition research had a fundamental role in agricultural development and improvement of nutrition in India. It was not surprising, therefore, to find the Commission recommending in their report the establishment of a

central institute for nutrition research. McCarrison took advantage of this recommendation and proposed to the Indian Research Fund Association that the unit under him be recognised as equivalent to a central institution. From 1929 onwards the unit at Coonoor was known as the Nutrition Research Laboratories and was the only institution in India devoted to researches in human nutrition. Even today it is the only one of its kind.

There was, however, a growing realisation on the part of young Indian scientists that the science of nutrition offered a wide scope for fruitful research. As mentioned earlier, McCarrison's researches and fame had made that realisation possible. One finds from 1930 onwards an increasing number of scientific workers contributing their quota to the growing knowledge in nutrition. These workers are spread all over India and are carrying out their investigations in medical colleges, universities and research institutes. Here again, the debt that all these workers owe to the Indian Research Fund Association must be acknowledged. The Association financed and maintained the Nutrition Research Laboratories at Coonoor. At the same time it afforded generous assistance to deserving individual workers. This financial support was essential, since in its absence no work could have been possible. In most teaching institutions in the country funds were usually not made available for research. Although the situation has improved to a certain extent it has not altogether eased. The Indian Research Fund Association, now the Indian Council of Medical Research, still spends annually an appreciable proportion of its total income in financing nutrition research.

A significant development in June 1936 which has influenced the progress of nutrition research in India was the formation of a Nutrition Advisory Committee by the Indian Research Fund Association. The primary function of this Committee has been to advise the Scientific Advisory Board of the Association on the various research proposals and programmes submitted to the latter for financial support. During the fifteen years of its existence, the Committee has performed several other functions as well. The Government of India was quick to realise the importance of the assistance which the Committee could give in an advisory capacity in matters connected with public health nutrition. In 1937, the Government accorded recognition to the Committee which made it the National Nutrition Committee, a privileged position which the Nutrition Advisory Committee still enjoys. In its capacity as a national body the Committee has tried to review and co-ordinate work in public health nutrition carried out in various administrative units of India. The Committee took part in planning for postwar reconstruction which had become a feature of governmental activity during the latter half of World War II. It has also considered such practical problems as technique of diet surveys, methods for the evaluation of nutritional status, dietary allowances, etc., and has recommended programmes of investigations designed to yield scientific information on these intricate problems. The Committee has tried to co-ordinate nutrition researches in India and to prepare fresh plans. On the whole, its advice has been invaluable to the Indian Research Fund Association in its object of furtherance of research. The results of these researches have been published mostly in Indian journals,

although a few papers have been published abroad. It was felt that a time had arrived when a review of these researches would be of great use to scientific workers in India and abroad if the material published over several decades and in various journals was collected and presented in the form of a connected account.

## CHAPTER II

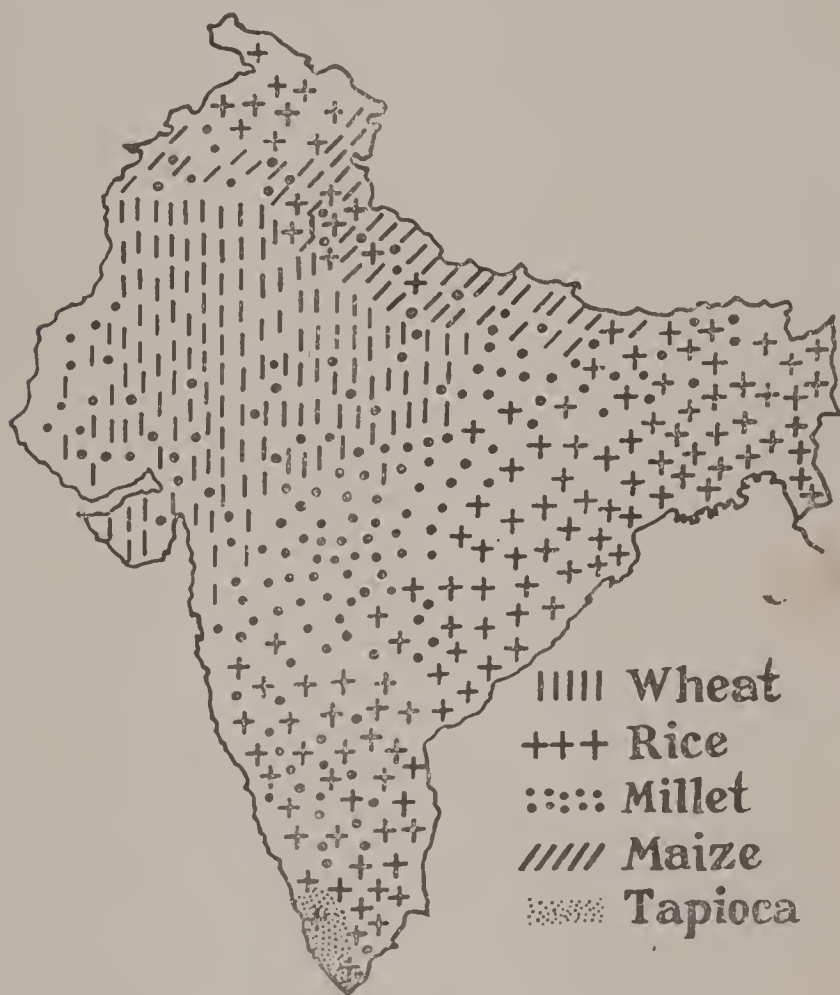
### INDIAN FOODSTUFFS—GENERAL

AN AMAZING VARIETY of foodstuffs is grown in India for human consumption. Considering the differences in soil, climate and rainfall that exist in different parts of the country, this must be considered inevitable. It is true that the production of foodstuffs is far below the maximum that can be reached if modern scientific methods were applied. The present relative food shortage is mainly due to continued adherence to old-fashioned agricultural practices, ignorance and illiteracy, which in turn engender and confirm superstitions; and above all this, there is the general poverty of the people. The fact must be faced that during the first half of this century much that could have been done was left unattempted. The state of planning in which the country indulged in the nineteen-forties only helped to show up the magnitude of the task and the colossal sums required to be spent on its accomplishment. It is worthwhile asking whether a fraction of those plans could not have been implemented gradually over a number of years. The fact that they were not even thought of or if conceived, the plans were shelved for no better reason than financial stringency shows an utter lack of foresight. It was inevitable, therefore, to find ourselves in a position today when the targets of production appear astronomically large and hence appear unattainable in the near future. Had it been otherwise India would not have found herself in this desperate position today. On the other hand, it is true that with the rate of growth of population being what it is,



India could never have expected to be self-sufficient. She could have produced, however, a greater proportion of her needs than she is doing today.

**Cereals:** The diet of Indians is primarily based on cereals from which 70 to 80 per cent of their energy requirements are met. In this they resemble other inhabitants of the tropical regions. Among the



## Geographical location of various staple food crops in India.

Figure I—Regions of Cereal Production in India  
 (Reproduced from Food and Nutrition)

important Indian cereals are rice, wheat, jowar (*Sorghum vulgare*), bajra (*Pennisetum typhoideum*), ragi (*Eleusine coracana*), maize (*Zea mais*) and barley (*Hordeum vulgare*). There are large tracts in India given over to the cultivation of only one type of cereal which is probably the result of the conditions determined by the soil, climate and monsoon. The accompanying map (Fig. 1) shows the distribution of some of the important crops in undivided India.

**Pulses :** In the Indian dietary, pulses play a very important role. These belong to the order leguminosae and there are a few varieties which are commonly used. They are Bengal gram (*Cicer arietinum*), red gram (*Cajanus indicus*), black gram (*Phaseolus mungo*), green gram (*Phaseolus radiatus*) and lentil (*Lens esculenta*). In most parts of India, one or the other of these pulses is incorporated in the diets usually in the form of thick cooked soup called *dahl*, spiced or unspiced. In addition to the above, a few other legumes like cow gram (*Vigna catiang*), horse gram (*Dolichos biflorus*), peas (*Pisum sativum*) and khesari gram (*Lathyrus sativus*) are used in certain regions.

The term gram refers to the dried seeds in husk and pulse to the decorticated seed in which the two cotyledons have separated. These foodstuffs are of importance in Indian dietaries for they are rich protein sources, their protein content varying from 20 to 25 per cent on the air dry weight.

**Vegetables :** Among the well-known varieties of vegetables there are about forty different leafy vegetables, 12 of root and tuber vegetables and over 40 other vegetables which are consumed in India. Several of them are common throughout the country, although the seasons when they are available differ from place to place. In most places green leafy

vegetables can be had most times of the year although in areas with extremes of climate, they become scarce during the summer season. On the other hand, fruit vegetables are scarce during monsoon.

The green leafy vegetables could be a most useful source of carotene, vitamin C and calcium and phosphorus in particular. Unfortunately their use is not so popular as one should like and it is ironic that either through ignorance or prejudice or probably both, these cheap foodstuffs are not made sufficient use of by the people.

**Meat, Fish and Eggs:** The only justification for lumping together of these three animal foods is that they are equally scarce. A large proportion of the population would readily consume any of them provided they were available and sufficiently low priced to enable the people to find them within their means. On the other hand, there is a large section of the Indian population which has religious objections to meat of one kind or other or against any kind of animal food with the exception of milk and milk products. The Hindus object to beef; Muslims object to pork. Sometimes these religious objections go much deeper. Sikhs would eat meat if the animals were killed in one way and Muslims if the other way. These objections and prejudices have unnecessarily multiplied the difficulties of the already complicated task of finding enough meat.

India has a large coast line in relation to her size and the potentiality of fish for food has not yet been fully exploited. Unfortunately, the same considerations which have been operative in agriculture have prevented any advances being made in exploiting this important source of food to the full. Most of the people along the coastline and in the interior too

would eat fish if it is made available to them in an unspoilt condition. The Bengalee is an exception. He is partial to fresh-water fish. The peculiar conditions in Bengal originating in the fact that Bengal is a marshy place studded with ponds as stars in heaven and that large rivers like Ganga and Brahmaputra wend their long course through it have made the Bengalee fresh-water fish-minded. Unfortunately the production of fresh-water fish is also low in relation to the needs, although it is true that it can be considerably improved with a little more planning and care.

Eggs are mostly obtained from peasants and small farmers who keep poultry. In keeping with the other standards of food production poultry is also largely untouched by modern science. Hen's eggs are of course the most common commodity, but in some parts of India a considerable number of duck's eggs also find their way into the market.

**Milk and Milk Products:** Milk is another important foodstuff which is produced in quantities far below the requirements. The estimated output in the year 1940 was about 5 ounces per capita. Recent estimates have shown that the total production per capita has remained practically unchanged. In actual practice the per capita consumption must be much less. A very large proportion of milk is converted into various other products for human consumption. A detailed description of these products will be given later.

In India two types of milk are of nutritional importance, cow milk and buffalo milk. The relative proportions of these milks produced according to a recent estimate are roughly 1 : 1. Goat's milk is only rarely used. The average composition (Schneider *et*

*al* 1948, and Sukhatme *et al* 1948) of the two important types is given in table I.

TABLE I  
COMPOSITION OF COW AND BUFFALO MILK

		Cow	Buffalo
Specific gravity	...	1.0307	1.0314
Total solids	... %	13.45	16.77
Solids-not-fat	... „	8.46	9.32
Fat	... „	4.97	7.45
Lactose	... „	4.59	4.88
Protein	... „	3.18	3.78
Non-protein-nitrogen	... „	0.054	0.051
Ash	... „	0.743	0.780
Calcium	... „	0.205	0.259
Phosphorus	... „	0.160	0.196
Chlorine	... „	0.088	0.066

It will be clear from the table that the fat content of cow milk is higher than that reported from Europe and America. The fat in buffalo milk is still higher, sometimes reaching as high a figure as 10 per cent in individual samples. Another notable difference lies in the vitamin A value of the two milks. Cow milk contains both preformed vitamin A and carotene whereas buffalo milk contains only the former.

**Unusual foodstuffs:** Apart from the broad classes of foodstuffs mentioned above, a considerable number of less known foodstuffs are consumed in



circumscribed regions. Our knowledge about several of them is incomplete. Mitra and Mittra (1941, 1942, 1943 and 1945) who have made a special study of these in the province of Bihar have listed quite a good few. In other parts of India too several articles are locally consumed which are not commonly used by the rest of the people. These foodstuffs may be of animal origin such as red ants and white ants, bull frogs, woodsand piper, snake and crocodile meat, etc. Unusual foods of vegetable origin are numerous, extending from less known cereals like *Panicum milliare*, *Setaria italica*, *Paspalum scorbiculatum*, etc., to different varieties of beans, leafy vegetables, fruits, roots and tubers, etc. Some of these are cultivated for human consumption and hence are of local importance. A detailed study of such foods from the standpoint of nutrition and the extent to which and the regions in which they are consumed, is certainly worth undertaking.

**Beverages — Alcoholic and Nonalcoholic:** Beverages contribute appreciably to the nutrition of certain sections of Indians. This is true of both the alcoholic and nonalcoholic varieties.

The consumption of alcoholic beverages has been referred to in Vedic literature. It is believed that 'soma' of the ancient Aryans was an alcoholic drink, although the raw material from which it was made is not known. In ancient Hindu writings up to 5th century A.D. mention has been made of the various alcoholic beverages prepared from molasses, mahua flowers, rice and barley. Even to the present day the art of preparing fermented liquor from several natural products is practised by tribal people in the plains and in mountainous regions. The reason why the fermentation industry in India was not placed on an

organised basis may lie in the fact that Hindus generally considered drink as a social evil. It is understood that according to the tenets of the Islamic religion also, drink is taboo. Necessarily, therefore, the preparation and consumption of alcoholic beverages in India has been confined to the lower strata of society. The advent of the British and the consolidation of their rule in India brought with them the foreign fermented and distilled liquors and it cannot be denied that a certain amount of liking for beer, wines and spirits has developed among the Indian public.

In India, local sources such as cereal grains like rice, wheat and barley, sugar cane molasses, juices from coconut and toddy palms and sugar rich mahua flowers (*Bassia latifolia*) have been used for the preparation of fermented and distilled liquors. Among these the consumption of *toddy* (fermented palm juice) and *arack* (distilled liquors flavoured with essences) has spread on a vast scale in towns and large villages. As against these, certain smaller communities consume the locally fermented preparations from rice and other cereals. Chopra and Chopra (1933) describe the practice of preparing such drinks with the help of a mixed inoculum consisting of yeasts and moulds. Ray (1932) refers to a 'national' drink of the hill folk of Darjeeling in the Himalayas prepared by fermenting cooked ragi (*Eleusine coracana*). Tribal people of the Chota Nagpur plateau in Bihar consume a drink known as 'Pachwai'. Its spread to neighbouring Bengal has been referred to by Neogi (1936). Chopra, Chopra and Chopra (1942) have made a historical survey of the alcoholic beverages in India and their article should be consulted for further detail. One observation in connection with certain tribal communities

is worth recording. When they drink the fermented (but not distilled) liquor they do not reject the residue of the cereal that is left behind. It is also consumed, so that this practice probably ensures the full utilisation of the food value of the fermented material.

Among the nonalcoholic beverages, tea and coffee are the most popular. Whereas consumption of coffee is restricted mainly to South India, tea is enjoying increasing popularity in the rest of the country. Tea habit has increased enormously in extent during the memory of the author. About twenty-five years ago the use of tea as a beverage was almost unknown in large areas of the Indian sub-continent, whereas today tea has firmly established itself in an increasing number of homes. In India tea and coffee are usually taken with milk and sugar.

Dilute butter milk is another beverage which enjoys a good deal of popularity in certain parts of India. In the Punjab and United Provinces, particularly during summer months, butter milk is relished as a cool and refreshing drink.

**Chemical Composition of Foodstuffs:** Several investigators in India have interested themselves in studying the chemical composition of Indian foodstuffs. A notable contribution in this respect has been made by the Nutrition Research Laboratories, Coonoor, where the work of Ranganathan, Sundararajan and Swaminathan (1937) led to over 200 foods being analysed. The collected results were incorporated in 1938 in Health Bulletin No. 23 entitled 'The Nutritive Value of Indian Foods and the Planning of Satisfactory Diets', published by the Government of India. This bulletin has gone through four editions during the last twelve years and has proved extremely useful. Among other workers who also studied the

composition of Indian foods can be mentioned Basu at Dacca, Guha at Calcutta and Mitra at Patna.

It was only natural that in the beginning much attention was paid solely to moisture, fat, protein, carbohydrates, crude fibre, and mineral content of these foodstuffs. Lately, however, quite a good deal of attention has been devoted to the study of vitamin contents as well. The work of Swaminathan, Bhagvat, Ahmad, Guha, De, etc., has contributed a great deal to our knowledge in this respect. The results of the later work have been incorporated in the latest edition of Health Bulletin No. 23. There are still numerous gaps, however, which will have to be filled before the information on most of the foodstuffs can be considered complete.

The above is a general account of foodstuffs. Some of them are so important from the standpoint of nutrition in India that it is worthwhile going into greater details regarding their uses and nutritive value. Separate chapters will, therefore, be devoted to rice and other cereals, pulses, milk and milk products, etc., whereas some others will be treated in less detail.

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## CHAPTER III

### RICE

RICE FORMS the staple food of more than half the population of India. Its annual production in undivided India was estimated to be about 29 million tons. The chief areas of production were Assam, Bengal, South India, West Coast of India, and certain smaller areas in the remaining provinces of the country. After August 1947, subsequent to the partition of the country, the annual production has been estimated to be in the neighbourhood of 20 million tons. According to agricultural experts, about 200 varieties of rice are cultivated in India.

In days when food shortage did not exist, freshly harvested paddy was stored for allowing the rice to mature, the cultivator storing his own paddy for family consumption. The people liked rice which had been prepared from paddy stored for six months to a year. Nowadays, however, the storage of paddy is limited and fresh harvests have to be brought to the market either directly or through Government procurement schemes.

The varietal difference in the chemical composition have not been carefully studied. It is, however, known that proteins in rice may vary from 6 to 9 per cent,  $P_2O_5$  0.06 to 0.12 per cent and  $CaO$ , 0.06 to 0.11 per cent (Sreenivasan 1939). These are fairly large variations; similar differences may exist in the amount of thiamine, but it is safe to say that no attempt has been made to breed and propagate the nutritionally preferable varieties of rice for agricultural production. Sen (1916) has given analytical

figures for Bihar rice. Basu and Sarkar (1935) have analysed 18 varieties of Bengal rice, 16 of which were pure line strains and 2 hybrids. Chitre, Desai and Bharani (1948) have studied 36 pure bred strains from Bombay State for their proximate principles, minerals and niacin and carotene content. Sadasivan and Sreenivasan (1938) report analytical figures for proximate principles and minerals for 22 varieties of pure bred strains from Madras state. The influence of cultural practices on the nutritive value of rice has received even less attention. McCarrison (1928) observed that rice grown by dry crop method was superior in nutritive value to rice raised as a wet crop. On the other hand, Sreenivasan and Sadasivan (1942) reported that dry cultivated rice was least effective in promoting growth of young albino rats when supplied as sole source of protein and minerals, and wet cultivated transplanted rice most effective; the irrigated broadcast rice had given an intermediate growth response. These authors also report a higher protein and mineral content in irrigated rice, than in dry cultivated rice. Earlier experiments on the effect of manurial treatment reported by Sadasivan and Sreenivasan (*loc. cit.*) do not indicate appreciable alterations in the composition of rice with respect to protein, calcium and phosphorus. It is evident that further work is needed on this problem particularly from the standpoint of the effects which cultural differences have on the vitamin content of rice.

**The Chemical Composition of Rice:** The results of analyses carried out by different investigators mentioned above have been incorporated in Table II. This is not an exhaustive list; there are other analyses available too, but out of the five references quoted in the Table, four are of recent

TABLE II—COMPOSITION OF INDIAN RICE

RICE Varieties and Source	Number and samples	Moisture %	Protein %	Ether Ex- tractives %	Carbo- hydrate %	Crude Fibre %	CaO %	P <sub>2</sub> O <sub>5</sub> %	Fe <sub>2</sub> O <sub>3</sub> %
<b>BENGAL</b>									
<i>Aus</i> —Raw	8	10.3	7.18	1.58	79.17	0.59	0.021	0.76	0.016
Husked Rice	8	11.2	6.76	1.04	79.98	0.22	0.017	0.45	0.013
Polished Rice									
<i>Aus</i> —Parboiled	8	9.94	7.69	1.53	79.41	0.21	0.019	0.71	0.015
Husked Rice									
<i>Aman</i> —Raw									
Husked Rice	10	9.95	7.71	2.19	78.32	0.67	0.06	0.60	0.014
Polished Rice	10	10.9	7.12	1.64	79.12	0.27	0.018	0.45	0.013
<i>Aman</i> —Parboiled									
Husked Rice	10	8.64	7.79	2.05	79.20	0.26	0.060	0.58	0.014
<b>MADRAS</b>									
<i>Pure Bred Strains</i>									
Raw-Husked Rice	22	10.9	6.65	2.38	77.4	0.54	0.07	0.75	—
Raw Polished Rice	22	11.6	5.65	0.56	81.5	0.23	0.03	0.28	—
Parboiled Husked Rice ...	12	10.4	6.61	—	—	—	—	0.62	—
Parboiled Polished Rice...	12	10.2	5.68	—	—	—	—	9.30	—
<b>BOMBAY</b>									
<i>Pure Bred Strains</i>									
Raw Husked Rice	36	10.95	5.98	2.08	79.63	—	0.148	0.56	—
<b>BIHAR</b>									
Raw Husked Rice	—	11.95	7.48	2.36	75.86	0.76	—	0.70	—
Raw Polished Rice	—	10.89	7.25	0.88	79.99	0.20	—	0.36	—
<b>UTTAR PRADESH</b>									
Raw Husked	12	—	8.4	—	—	—	—	—	—
<b>ORISSA</b>									
Raw Husked	7	11.94	7.78	—	—	—	0.034	0.68	—

origin and include the analysis of rice samples obtained from practically the major portion of rice growing regions. It is to be noticed that the recorded average figures for protein in rice from Madras and Bombay States are lower than those found for Bihar and Bengal varieties. In other respects, there seems to be little difference.

**Vitamins in Rice:** There have been even fewer investigations in India on the vitamin content of rice. Although a few coloured varieties of rice may show traces of carotenoids, rice is of importance only from the standpoint of its contribution to the diet of vitamins of the B complex, particularly vitamin B<sub>1</sub>. Table III summarises the available information on the vitamin content of Indian rice.

TABLE III—VITAMINS B IN RICE

Variety and number of samples		Thiamine μg per gm.	Riboflavin μg per gm.	Niacin μg per gm.	Pyridoxine μg per gm.
Brown (5)	...	2.9	—	—	—
Brown (1)	...	4.0	—	—	—
Handpounded (8)	...	2.4	—	24.0	—
Machine milled (8)	...	1.0	—	16.0	—
Brown (11)	...	2.1 to 4.8	—	—	—
Brown	...	—	—	46	6.9
Handpounded	...	—	—	24	—
Milled	...	—	0.3	12	2.3

Numbers in parentheses indicate the number of samples analysed.

The above will reveal the vast gap which exists in our knowledge regarding the vitamin content of different varieties of rice.

**Storage of Rice:** Rice is usually stored in husk for varying lengths of time. Husked rice is difficult to store, as it is liable to insect and fungal attack during storage. On the other hand, milled rice keeps better than merely husked rice. Rice obtained from freshly harvested paddy cooks into a glutinous mass. There is no doubt that during storage the cooking quality improves. Rao (1938) ascribes this improvement to a completion of the change taking place in starch due to slow dehydration occurring during storage. Sreenivasan (1939) finds that well stored grains take up about twice as much water during cooking as unstored grains, the former proportionately swells much more and gives a cooked product as discrete grains. Stored rice is also more easily digestible than fresh harvested rice although the *in vitro* digestibility of pure starches prepared from two grains is not appreciably different. Thiamine content of rice as affected by storage has been investigated, but systematic investigations controlling temperature, humidity and time are lacking. Passmore and Sundararajan (1941) found negligible losses in thiamine after three months' underground storage of paddy. Rao, Ramachandran and Rau (1942) also found no thiamine loss in samples of paddy stored in stoppered bottles for three years. On the other hand, Chitre (1949) reports the almost complete loss of thiamine in bottle-stored paddy. The observations of Kik and Williams (1945) in the U. S. A. are interesting. They observed a loss of 8 to 15 per cent in thiamine after storage for nine months at 84°F whereas the losses in cold storage for 2½ years were negligible.

Although the consumer is unaware of the changes taking place on storage, he prefers stored old rice to



the freshly harvested produce for the reason that the former cooks well.

**Milling:** Rice is usually consumed after subjecting it to either hand-pounding or milling. The practice of hand-pounding was, before the installation of mechanical devices, the sole means of treating rice for removing the bran layers. Machine-milling, since its introduction in India, has become of increasing importance in rice industry. It has gradually but firmly ousted hand-milling to a considerable extent.

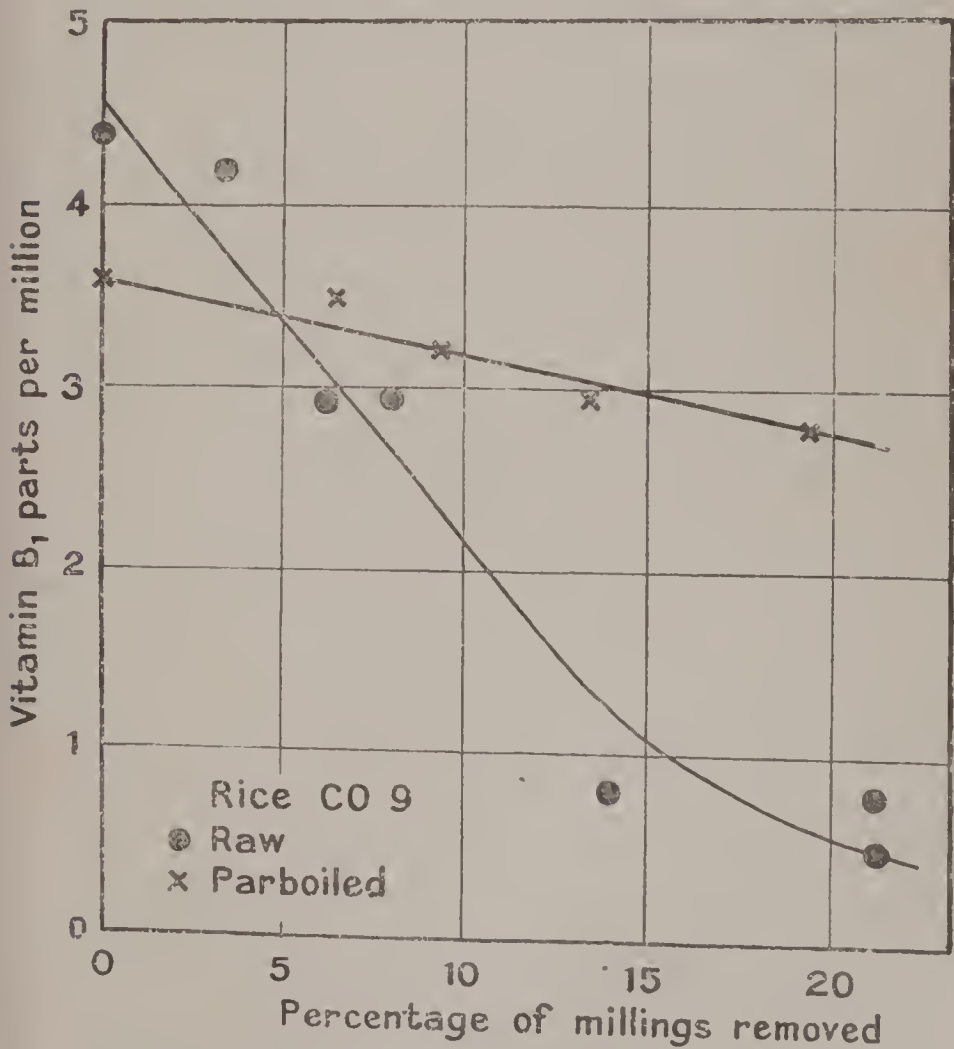


Figure II—Loss of thiamine on milling of raw and parboiled rice.

It is to be noted, however, that in villages of India, rice is still hand-pounded, but the rice prepared for the market is usually machine-milled.

When rice is dehusked, the shell is removed leaving behind the embryo attached to the rest of the grain. This attachment is, however, not very strong. In hand-pounding as well as milling the embryo is the first to be detached and simultaneously the bran layers continue to be removed, the removal being in proportion to the degree of milling. Approximately,

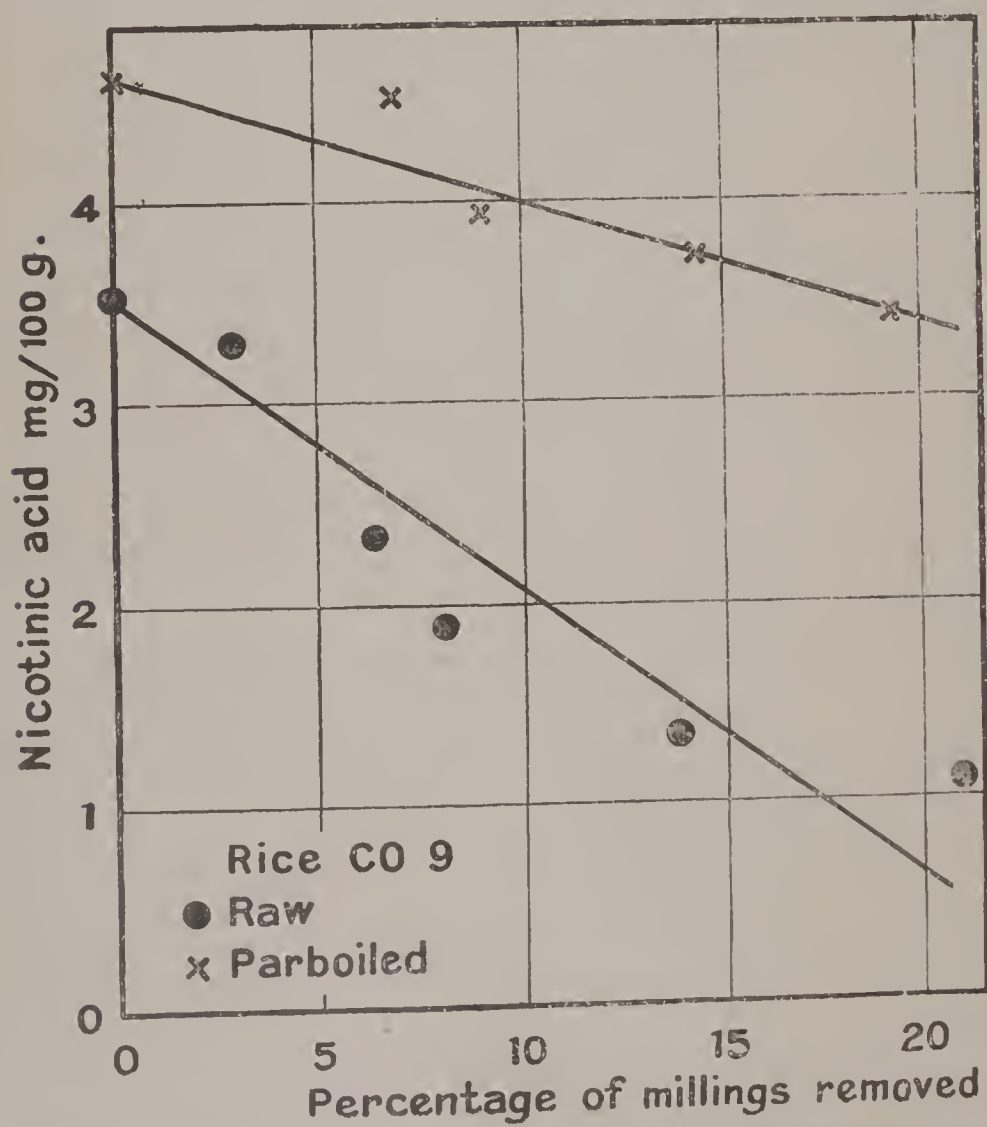


Figure III—Loss of nicotinic acid on milling of raw and parboiled rice.

16 to 28 per cent nitrogen and 80 to 84 per cent phosphorus were found to be removed in two varieties of rice when they were subjected to experimental milling so as to remove 24 per cent of the grain weight. This loss is not inconsiderable but the losses of B vitamins are more serious. Figures II and III show the extent of removal of thiamine and nicotinic acid when rice is milled.

The inability of rice-eating people to supplement their diets with nutritious foodstuffs owing to the widespread poverty makes the milling of rice a positive public health danger.

The time honoured but slow method of hand-pounding is less destructive in that it gives nutritionally a better product. The usual practice is to pound rice with a long wooden pestle in an iron, wooden or stone mortar. The removal of bran layers is imperfect and hence the losses of nutrients also are less complete. The following table (Table IV) based on figures reported by Aykroyd *et al* (*loc. cit.*) will illustrate the difference in the thiamine content of hand-pounded and machine-milled rice.

TABLE IV—THE EFFECT OF PROCESSING ON THIAMINE CONTENT OF RICE

Variety	Raw			Parboiled	
	Hulled	Hand-Pounded	Machine-milled	Hand-Pounded	Machine-milled
$\mu\text{g}$ thiamine per gram.					
Vadan Samba ...	2.5	2.1	0.6	2.1	2.1
C 0.9 ...	3.7	2.9	1.3	2.4	2.0
ADT-111 ...	2.9	2.1	1.3	2.4	1.6
GEB-24 ...	2.9	2.1	1.3	2.0	1.7

Thus in machine-milling, up to 75 per cent of thiamine may be lost in the bran; higher losses are not unknown. In hand-pounding, on the other hand, losses average about 25 per cent. There is no doubt, therefore, that hand-pounding of rice is better than milling. Now that milling of rice has replaced hand-pounding to a large extent, reversion to the old methods is impracticable. During the war, various provincial governments in India had enacted laws with a view to obtaining undermilled rice, their main object in doing so was to produce more rice for the market by saving bran losses incurred in milling. As a result of this policy rice of indifferent appearance, varying cooking quality and poor keeping quality has been offered for consumption. The undermilled rice does not store well. The small amounts of fat in the grain tend to get rancid and spoil the quality of the product. Further, owing to the high humidity which exists in certain times of the year in most of the rice producing and consuming regions it falls an easy prey to bacterial and fungal attack. The restriction on milling of rice raises the problem of the high phytin content of rice interfering with the absorption of calcium and iron. All these disadvantages due to undermilling assume importance when rice has to be supplied to urban areas. It is obvious therefore, that undermilling of rice to a controlled degree is only one of the methods of producing rice of high nutritional quality.

**Parboiling of Rice:** A very large proportion of paddy in Madras, Bengal and on the west coast is subjected to a process known as parboiling. This consists in soaking paddy in cold or warm water for varying lengths of time, followed by steaming till the grain becomes soft and partly or wholly cooked.

The excess water is drained off and paddy is spread out to dry. Dried parboiled paddy is then hulled giving a product known as parboiled rice. The grain is slightly coloured, harder than the original rice grain and may possess an odour which varies from a faint nondescript type to a definite offensive one. Parboiled rice is subjected to the usual process of hand-pounding or milling. When cooked, it gives a product in which the cooked grain is discrete and rather hard to the feel.

The practice of parboiling is old and seems to have changed little with the passage of time. The methods are entirely empirical and differences may be found in (a) time of soaking, (b) temperature of steep water, (c) renewal or otherwise of steep water and (d) period of steaming. The final stage, i.e., drying in sun, is the most variable and hence defective since it depends upon the temperature and humidity of the surrounding atmosphere as well as air movement.

The method of parboiling has received some attention from several investigators. As yet, however, it must be admitted that these studies have made little impression on the age old methods which are still being practised in the production of parboiled rice. Aykroyd (1932) demonstrated by rat experiments that parboiled rice retains a considerable amount of vitamin B<sub>1</sub> even when highly milled. Sreenivasan (1936) studied parboiling on a laboratory scale by controlling the several variables in the process referred to above. He found that the yield of rice did not differ with different treatments in which the temperature of steep water was varied, as also the period of steeping from 2 to 72 hours and steaming at atmospheric pressure to 15 lbs up to 25 minutes. He noticed a slight enrichment (2 to 3 per cent) of



the kernel with nitrogen which he ascribed to the fact that parboiling probably rendered the outer coatings of rice kernel tough and therefore less liable to be lost in hulling. The smell of parboiled rice was found to depend upon the extent of fermentation which took place when rice was allowed to soak at room temperatures from 48 to 72 hours, particularly if the steep water was unchanged.

Although parboiled rice is slightly richer in nitrogen and phosphorus, the losses in these two elements are proportionately the same as in raw rice when subjected to an equal degree of milling. Thus in this respect milling seems to have a different effect to that found for vitamins. The effect of milling of parboiled rice on its thiamine and nicotinic acid content has been illustrated in Figures II and III. Aykroyd (*loc. cit.*) suggested that during parboiling the vitamins from the outer coats of rice kernel penetrated into the grain and were more evenly distributed thus accounting for their greater retention in the parboiled milled rice.

This explanation held the ground till the detailed researches of Hinton (1948) threw additional light on the events responsible for vitamin distribution in rice grain during parboiling. Nicholls (1947) had earlier suggested that in parboiled rice the scutellum, which was richer in thiamine than the endosperm, was more firmly attached to the grain and less liable to be removed by milling. Hinton demonstrated that a redistribution of thiamine from germ, scutellum and aleurone layers took place during parboiling with the result that the endosperm became richer in thiamine than that of raw rice. He also confirmed Nicholls' suggestion that the germ and scutellum attained a firmer attachment to the rest of the grain during parboiling.

According to Hinton, steeping for four hours at  $75^{\circ}\text{C}$  achieves the object of vitamin redistribution without causing much destruction of thiamine and that steaming is no longer necessary. Rao (1949) has developed a method in which paddy is steeped for two hours in a 0.2 per cent solution of calcium chloride at  $70^{\circ}\text{C}$  and dried in mechanical driers. The product obtained is light-coloured and odourless and is richer in calcium than raw rice.

Thus it will be clear that it is not impossible to obtain an acceptable and at the same time nutritionally desirable product by parboiling. The pressing need of the moment is to introduce practices based on scientific knowledge in actual manufacturing practice. During World War II, the possibility of installing a Huzenlaub plant for rice 'conversion' was explored but on mature consideration it was found that although the process was undeniably a good one, it was not suitable to the peculiar conditions in India governing rice production and marketing. That does not mean, however, that current practices cannot be modified. Improvements can be introduced provided the rice-milling industry is willing to co-operate. It is time that it did.

**Other methods of processing rice and paddy:** There are some methods in common use throughout India according to which paddy is treated so as to give products ready to be consumed without further cooking. Four such products are known which depend upon the procedure adopted as described below.

1. Paddy is wetted with a little water. It is then mixed with about four times its volume of pre-heated sand in a frying pan kept on the open fire, the temperature of the sand being about  $230^{\circ} - 240^{\circ}\text{C}$ . Paddy

is rapidly stirred with an iron ladle and after two to three minutes it is thrown over a wire sieve which removes the sand leaving behind parched rice which has swollen and burst out of the cracked hull during parching. The temperature of the mass immediately after the removal of sand is between  $130^{\circ}$  -  $140^{\circ}\text{C}$ . The sudden rise of temperature from that of the room to  $130^{\circ}\text{C}$  causes a very quick evaporation of moisture contained in the grain and the latter opens out, and gives an appearance as if something had mildly exploded inside the cereal. The final product which has different names in local vernaculars, is very tasty and is usually used as food for convalescents.

2. Paddy is steeped in warm water, the temperature of which to begin with is  $60^{\circ}$ — $70^{\circ}\text{C}$ . After steeping overnight during which time the mixture has cooled down to room temperature, water is drained off. Small amounts of steeped paddy are heated in a shallow pan till the husk begins to crack, then transferred to a wooden mortar and pounded by means of a wooden pestle. The grain is beaten flat and in the same operation is dehusked. The product is then dried. An elongated, flat grain with partially broken margins is obtained. It can be eaten as such or after frying in oil or ghee and mixed with other spices.

3. The beaten flat rice obtained in the process described in the last paragraph is parched with pre-heated sand. The flat grain then swells and assumes a 'roasted' taste and flavour.

4. Paddy is boiled in water, dried and dehusked. The grain is then parched as already described.

The above mentioned products do not form part of regular meals, but as constituents of between-meal snacks they enjoy great popularity as sweet or savoury

preparations mostly the latter. These procedures while enhancing the palatability of the product do not adversely affect the digestibility and biological value of rice proteins; on the other hand, slight increases in both have been observed by Acharya, Niyogi and Patwardhan (1942).

**Nutritive Value of Rice:** In dietaries based on rice, the latter forms a major source of proteins and thiamine apart from its contribution of starch in which it is exceedingly rich. Hence the digestibility of rice protein and its thiamine content are extremely important from nutritional standpoint. Several reports of investigations in India are available which show that the digestibility of rice protein is well over 90 per cent and its biological value is also comparatively high. Using the balance sheet method at 5 per cent protein level and rats as experimental animals, Swaminathan (1937) reported for rice protein a biological value of 80. Similar figures for raw, polished and parboiled rice are given by Basu and Basak (1937) and Acharya, Niyogi and Patwardhan (1942). Working with 6 adult human subjects, Mitra and Verma (1948) found a biological value of 67 for rice in a diet containing nearly 20 ounces or more of rice and approximately three to four ounces of pulses per day. The above figure is identical with that obtained by Basu, Basak and De (1941) on one subject.

The contribution rendered by rice to dietary thiamine is conditioned in India by a number of circumstances. It depends upon whether one consumes raw or parboiled and hand-pounded or milled-rice. The effect of parboiling and milling has been discussed earlier and it has been shown that an improved method of parboiling would ensure a product

with a fairly high thiamine content and that losses on milling of such rice would be comparatively small, so that the public demand for lightly coloured milled-rice could be met without unduly sacrificing the quality of rice.

There is another and a very important step where rice loses quite a good deal of thiamine before it is finally eaten. That step consists of preparation of cooked rice. The common practice of cooking rice in India is described below to enable one to understand the various stages where such losses might occur. Rice as purchased is first cleaned of broken grains and foreign matter. It is then washed repeatedly with plenty of water. At least three washings are common while more are not infrequent. The wash water is allowed to drain and the wet rice is stirred in into a pot containing water—just brought to boil. The pot is heated on open fire. As the water boils vigorously the rice grain gradually swells. At the same time it loses part of its starch and some nitrogenous material which forms a viscous gelatinous fluid. When the rice grain has softened and imbibed the maximum amount of water, the excess of the water is drained off by tilting the vessel. The final step consists in allowing the rice in a covered pot to be fully cooked by the steam from residual water. During this time only gentle heat is necessary. Well cooked rice should be soft to the core with each cooked grain remaining discrete.

Raw milled rice may lose up to 60 per cent and parboiled rice 10 per cent during washing. Larger the number of washings, greater the loss. The first washing is responsible for 40 - 50 per cent loss of thiamine in rice (Swaminathan 1942), subsequent washings removing much less. The second stage



where losses may occur is when excess water after the rice is almost completely cooked is drained off. Table V illustrating losses during cooking is based on the figures reported by Aykroyd *et al* (1940) as a result of controlled experiments in the laboratory.

TABLE V  
LOSSES OF THIAMINE IN COOKING OF RICE

Rice		Thiamine in rice $\mu\text{g./gm.}$		
		Uncooked	Washed and Cooked	Loss %
<b>Co-5</b>				
Raw milled	...	0.8	0.2	75
Parboiled milled	...	1.9	0.9	53
<b>Co-2</b>				
Raw milled	...	0.9	0.1	89
Parboiled milled	...	2.1	1.0	52
<b>Co-7</b>				
Raw milled	...	0.9	0.3	67
Parboiled milled	...	2.6	1.1	58

It must be mentioned that in actual practice the losses in thiamine must be more than the above figures suggest, for the simple reason that there is no control on the amount of water used for washing or for cooking. There are, however, certain regions in India where cooking losses are somewhat less, for there rice is cooked in just as much water as it would imbibe. This method requires a little skill and experience of the cooking quality of rice.

It may be of interest to mention that work done later in the U. S. A. by Kik and Williams confirmed most of the findings of workers in India on the effect of processing, washing and cooking of rice on its thiamine content.

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## CHAPTER IV

### WHEAT AND OTHER CEREALS

THE AMOUNT OF WORK done in India on wheat is much too little compared to the detailed investigations on rice already described. This difference is noteworthy for apart from rice wheat also forms an important cereal in Indian diets. Punjab, Delhi and a major portion of Uttar Pradesh are the main wheat producing areas and also those where wheat forms the staple cereal. Parts of Bihar, Central India and Central Provinces are other regions where also people eat wheat. Rajasthan, Sourashtra, Berar, Hyderabad, Bombay State and Mysore are the large millet producing parts of the country. In these places, *jowar* (*Sorghum vulgare*), *bajra* (*Pennisetum typhoideum*), and *ragi* (*Eleusine coracana*) are the chief millets.

One reason which, according to the author, may possibly explain the lack of interest on the part of investigators may be that these food grains are very much less subjected to processing as compared to rice. Sporadic attempts to study the chemical composition of these food grains have been made; their results will be referred to under each individual food grain.

**Wheat:** The average composition of Indian wheats can be found in Health Bulletin No. 23 and in a report by Mitra and Mitra (1941). In India the bulk of wheat is consumed as *atta* which is merely whole wheat ground to flour and used as such. The difference in the composition of the two is practically nil. At least in some homes the flour may be passed through a coarse sieve to remove the fibre rich bran.

Several attempts to determine the amount thus removed have given the figure which lies between 5 to 7 per cent by weight. This means that Indians use for their daily bread a flour of extraction rate which varies from 93-95 per cent. Bread as is known in the West is consumed by a negligible proportion of the people, although it is true that its consumption shows signs of increase. It is the unleavened bread, or *chappatie* as it is known, that forms the commonest preparation of wheat flour, and to make good *chappaties* the refining of flour is not necessary. Even in *chappatie* making there are interesting variations which should be described briefly to give an idea of Indian food habits with regard to wheat.

There are three or four different methods which are in vogue in different parts of India. The simplest consists in mixing flour with the requisite quantity of water and kneading the mixture till the dough is obtained. The dough is allowed to stand for some time, e.g. half to one hour, and then given a second kneading, after which a small portion of the dough is rounded off between the palms of the hand and rolled on a wooden board with a rolling pin into a flat circular and thin (about  $1/16''$  to  $1/12''$  thick) *chappatie* which is then placed on a shallow pan on an open fire. One side of the *chappatie* gets baked in about two minutes when it is turned over and baked on the other side. After both sides are baked the *chappatie* is placed on fire direct and manipulated by hand or a pair of tongs when it separates in two layers and is ready for eating. There are minor variations in this general procedure resulting in thick or thin *chappaties*. In some places a little oil or ghee and salt are mixed with the dough. With this last procedure one can obtain *chappatie* with a slightly



open texture and which has much less tendency to harden when kept for 12 to 24 hours.

A second method consists in preparing the dough with a little oil or ghee and salt mixed with the flour and rolling the dough as above. A thin layer of oil or ghee is smeared on one side and the *chappatie* folded, another thin layer is smeared and a second fold given. This twice folded *chappatie* is rolled again and baked on a flat open pan. In this method the *chappatie* need not be placed on the open fire. It is ready to eat when both sides are baked on the pan itself; as a further refinement one can add a little ghee to the pan and turn the *chappatie* twice when a product known as *paratha* is obtained.

A third and important preparation is called *purie*. Small flat pieces of rolled *chappatie* 3 to 4 inches in diameter are deep fried in ghee over open fire. The product is known as *purie*. Recently, hydrogenated oils have been used for frying *puries*, but that is mainly due to the prohibitive cost of butter and ghee. *Purie* is the food of the rich. Poor and middle class people can afford it only on festive occasions; simple *chappatie* is the poor man's food.

**Proteins of Wheat:** Just as rice makes a major contribution of proteins in a rice eater's diet, wheat does it to a larger degree to a wheat eater's diet. The digestibility and biological value of wheat protein have been determined both in rats and human beings. Swaminathan (1937) found a biological value of 66 and digestibility of 93 per cent in rats at 5 per cent level. Basu (1946) gives figures for B.V. and D.C. as 53 and 77 respectively for one human subject and 60 and 81 for another. The work on human subjects has been possible only in the presence of small quantities of other proteins, a complication which did

not exist in the work on rats. It seems safe to conclude, however, that in mixed diets the biological value of wheat protein does not differ greatly from that of rice protein so far as the human subject is concerned. In rats, on the other hand, there seems to be some difference in favour of rice.

Other products of wheat such as white flour, patent flour, semolina, etc. are used in India for making various food preparations but their use is not so important quantitatively as that of *atta*.

**Jowar, Bajra and Ragi:** These millets are extensively consumed in India; in figure I are indicated the regions where they are produced and form the staple cereals of the local population. Jowar and bajra are mainly consumed in the form of unleavened bread made from the flour of the whole grain. Since these flours contain little gluten, the dough cannot be rolled into *chappaties*. The freshly made dough of jowar or bajra is beaten on a flat surface sprinkled with dry flour by gentle tapping with the palm of the hand and fingers with a slight rotating action. A round and somewhat thick *chappatie* results which is baked on a shallow pan heated on open fire. When both sides are suitably done the final baking is done on open fire directly. Sometimes the crushed millet can be cooked to give a porridge which is also not infrequently eaten at meals. Ragi is poor man's food and in many places is the cheapest cereal. The common method of cooking it is to make balls of moistened ragi flour and to steam them. They can then be eaten with other foods.

A certain amount of information is available about the nutritive value of protein and vitamins B content of these foodstuffs. The information about the biological value (B.V.) and digestibility coefficients of

total proteins of cereals as found by experiments on albino rats is given in Table VI. The balance sheet method shows that the biological value of jowar, bajra and ragi proteins compares favourably with rice protein. On the other hand, results of the growth method show that they are inferior to both rice and wheat proteins which gave values of 1.7 and 1.31 respectively in a comparable experiment carried out by the same author.

TABLE VI—THE DIGESTIBILITY COEFFICIENT AND BIOLOGICAL VALUE OF SOME CEREAL PROTEINS

Source of Protein	Level of Protein fed %	D. C.	B. V.	B. V. by growth method	Reference
Jowar ...	5	91	83 ✓	0.78	(1)
	5	91	83	—	(2)
Bajra ...	5	89	83	1.15	(1)
Ragi ...	5	80	89 ✓	0.71	(1)
	5	93	90	—	(2)
	5	78	91	—	(3)
Maize ...	5	80	60	—	(2)

(1) Swaminathan, M. (1937).

(2) Acharya, B N., Niyogi, S. P., and Patwardhan, V.N. (1942).

(3) Niyogi, S.P., Narayana, N., and Desai, B.G. (1934).

The results of estimations of vitamins in these foodstuffs are given in Table VII.

TABLE VII—VITAMINS B IN SOME CEREALS

Cereal	Thiamine $\mu\text{g./g.}$	Riboflavin $\mu\text{g./g.}$	Nicotinic Acid $\mu\text{g./g.}$	Pyridoxine $\mu\text{g./g.}$
Wheat	... 3.76 <sup>1</sup> 2.5 to 4.2 <sup>2</sup> 3.6 <sup>3</sup> 4.5 <sup>4</sup>	1.19 <sup>7</sup> 1.2 <sup>4</sup>	23.3 <sup>8</sup> 31.0 <sup>5</sup>	8.1 <sup>4</sup> 6.4 <sup>4</sup>
Jowar	... 3.5 <sup>1</sup> 3.8 to 4.9 <sup>2</sup> 4.1 <sup>4</sup>	Trace <sup>7</sup>	18.2 <sup>4</sup>	8.0 <sup>4</sup> 7.3 <sup>6</sup>
Bajra	... 3.0 <sup>8</sup> 3.8 <sup>4</sup>	Trace <sup>7</sup>	25 <sup>4</sup> 11.3 <sup>8</sup> 32 <sup>5</sup>	10.7 <sup>4</sup> 11.6 <sup>4</sup>
Ragi	... 4.2 <sup>1</sup> 2.7 to 4.0 <sup>2</sup> 4.2 <sup>4</sup>	11.3 <sup>7</sup> 0.5 <sup>4</sup>	11.6 <sup>5</sup>	4.3 <sup>6</sup>
Maize	... 4.2 <sup>3</sup> 4.1 <sup>4</sup>	3.1 <sup>7</sup>	14.8 <sup>4</sup> 11.8 <sup>8</sup> 2 <sup>5</sup>	

1. Passmore and Sundararajan (1941).
2. Rao *et al* (1942).
3. Bhagvat (1943).
4. Swaminathan (1938, 1940, 1942, 1946).

5. Giri and Naganna (1941).
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- (1942) — Ibid., **30.**, 23.
- (1946) — Ibid., **34.**, 289.
- Giri, K.V. and — Ibid., **29.**, 125.
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- Murthy, G.N. (1937)



## CHAPTER V

### PULSES AND LEGUMES

**Common Indian Pulses:** It has been mentioned earlier that pulses and legumes form important articles of diet in India. A study of Table VIII should make the reason obvious. Those Indians who have religious objections to using flesh foods derive a fair amount of protein from pulses and legumes. Others who have no such scruples and yet are denied the frequent use of animal foods through poverty also have recourse to these foodstuffs. Thus it seems that by experience the Indians, like other inhabitants of the tropical regions, have found a comparatively cheap source of protein to supplement their cereal rich diets. Although it is true that in the tropics and subtropical regions outside India leguminous foodstuffs are usually consumed, it is a moot question if pulses particularly are as commonly used elsewhere as they are in India.

In a report (1939) on Nutrition in the Colonial Empire (of Great Britain) reference has been made to the consumption of legumes of different species. In China and Japan soya bean is an important legume. In India, however, pulses are consumed on an extensive scale and therefore fall in the category of food crops which are next in importance to that of major cereals discussed earlier.

The pulses, pea (field) and beans like cow gram and horse gram have received some attention at the hands of investigators interested in nutrition. The crude protein content of these foodstuffs lies between 22 to 25 per cent calculated on air dry samples. The

TABLE VIII—PROTEIN AND VITAMIN CONTENT OF INDIAN PULSES AND LEGUMES

Legume— Common Vernacular Name	Botanical Name	Proteins			Vitamins				
		Total Protein value, %	Biological value, level	Digesti- bility %	Carotene I.U.—100g. (Vitamin A value)	Thiamine	Ribo- flavin $\mu$ g./g.	Nicotinic Acid	Pyrido- xine
Bengal Gram	<i>Cicer arietinum</i>	22.3	62	86	316	4.5	5	59	11
Black Gram	<i>Phaseolus mungo</i>	24.3	62	78	64	4.1	1.8	23	11.6
Green Gram	<i>Phaseolus radiatus</i>	24.6	51	86	158	7.5	5.2	17	10.8
Red Gram	<i>Cajanus indicus</i>	23.5	72	75	220	4.2	2.7	24	10
Lentil	<i>Lens esculenta</i>	25.3	41	88	450	4.1	—	15	—
Peas (dried)	<i>Pisum arvense</i>	23.5	48	91	—	4.4	—	10	—
Cow Gram	<i>Vigna catianga</i>	23.4	45	78	60	4.9	—	22	—
Horse Gram	<i>Dolichos biflorus</i>	22.5	66	73	119	4.2	—	18	—
Soya Bean	<i>Glycine hispida</i>	40.1	54	76	710	7.3	—	—	8.6

proteins vary in biological value and digestibility coefficient as determined on albino rats. In Table VIII are given the average values collected from the available data mainly derived from the researches of Swaminathan (1938), Basu and his associates (1936, 1937) and Niyogi, Narayana and Desai (1931, 1932).

Bengal gram, red gram, black gram, green gram and lentil are the five pulses most commonly used in that order although it would be difficult to prove it by actual figures for consumption. Bengal gram is used throughout India for making a variety of foods. The other grams are mainly used to make *dahl*, spiced or unspiced, although their incorporation in other types of foods is not uncommon. The digestibility of protein from these pulses at 10 per cent level was found to vary in rats from 75 to 90 per cent and the biological value from 41 to 73 per cent. From the nutritional standpoint, Bengal gram and red gram are superior to other pulses and grams and thus justify their great popularity.

In certain parts of India it is a common practice to soak the gram (with husk) and some beans also in water for 12 to 24 hours and then to tie the soaked grain in a wet cloth. The tied bundle is allowed to stand at room temperature for another 24-48 hours when most of the grains germinate. The easily removable husk is taken off and the germinated legume either eaten raw or consumed after cooking with spices. A reference to this wholesome practice will be made somewhat later.

Niyogi, Narayana and Desai (1931, 1932) have studied the globulins from a few of these pulses and have determined some of the important amino acids in the hydrolysed globulins. Their results are

TABLE IX—AMINO ACIDS IN GLOBULINS FROM EIGHT PULSES EXPRESSED AS PERCENTAGE OF ASH AND MOISTURE-FREE PROTEIN

Protein source Botanical name	Lysine	Histidine	Arginine	Cystine	Tyrosine	Tryptophane
<i>Cicer arietinum</i> ...	7.42	1.42	10.29	2.02	2.95	0.46
<i>Phaseolus mungo</i> ...	7.70	7.46	7.90	1.59	3.35	0.74
<i>Phaseolus radiatus</i> ...	6.16	3.46	6.37	3.51	3.63	0.84
<i>Cajanus indicus</i> ...	7.03	2.56	5.84	1.86	3.16	0.46
<i>Lens esculenta</i> ...	6.70	2.77	10.35	1.62	3.56	0.62
<i>Pisum arvense</i> ...	8.19	1.90	9.50	1.99	3.74	0.51
<i>Vigna catiāng</i> ...	5.96	2.21	7.45	1.89	3.74	0.59
<i>Dolichos biflorus</i> ...	8.15	2.38	6.00	1.24	4.01	1.02

From Niyogi, Narayana and Desai (1932)

summarised in Table IX. Although the information is incomplete, it is of certain value if it is remembered that the globulins isolated from the legumes mentioned in the table form a major fraction of the total proteins present in them. Desikachar and De (1947) have determined the cystine and methionine content of these pulses by the method of Evans (1945) and obtained values between 0.32 to 0.59 per cent and 0.14 to 0.43 per cent respectively on air dry samples. Basu, Nath and Mukherjee (1937) report that lentil is markedly deficient in cystine. The values reported by them do not differ much from those mentioned in Table IX and hence proteins of pulses in general can be considered as being poor sources of cystine and tryptophane.

Bhagvat and Sreenivasaya (1935) reported that pulses contained 10 to 55 per cent of its total nitrogen in a form which was dialysable and not precipitated by saturation with ammonium sulphate. They mention, however, that the fractions may consist of simple peptides and higher compounds intermediate between them and true proteins. Swaminathan (1938) who estimates the non-protein nitrogen by precipitation with copper hydroxide obtains a value of 2.9 to 13.4 per cent on the total nitrogen. This fraction which is true N.P.N. is probably due to the presence of nitrogenous compounds other than protein intermediate products.

In general, pulse proteins have a lower biological value than cereal proteins as judged by metabolic experiments on rats. The limiting factors, however, are not necessarily the same in both categories of proteins and hence a favourable supplementary effect of mixing the two can be expected. Some experiments of this type were carried out by Swaminathan



(1938) who claims to have obtained a supplementary effect when a cereal like ragi or rice was mixed with a pulse so that proteins derived from the two sources were in proportions of 50 : 50. His results, however, do not appear convincing. The B. V. of rice protein was 80 and that of Bengal gram 62, whereas that of the protein mixture from the two, 66. With the black gram protein of B. V. 62, mixture with rice gave a B. V. of 61. On the other hand, ragi proteins having a B. V. of 89, gave in mixture with Bengal gram and Black gram a B. V. of 76 for both. This value is approximately intermediate between the B.V. of ragi proteins and the two pulse proteins. From the results one can only conclude that rice somehow brings about a depression in the biological value of mixed proteins, whereas ragi proteins exert neither a beneficial nor an adverse effect. The results appear to be somewhat confusing and hence the conclusion of Swaminathan regarding the supplementary value of pulse and rice and ragi does not appear to be fully justified. This comment receives further support from the results reported by Mitra and Mittra (1947) who report that the B. V. of rice protein is depressed by pulses when added to give a proportion of 1 : 0.75 for rice and pulse proteins. A further increase in the proportion up to 1 : 1.25 does not make any significant difference in the B. V. of the mixture.

**Soya Bean :** Soya bean has excited a good deal of interest and controversy among the nutrition workers in India. It is an important crop in the neighbouring country, China, but in India its cultivation has not been pursued with vigour. Soya bean, however, is not altogether unknown in India. It has been cultivated to some extent in Bengal, Bihar and Orissa. In addition, it is grown in Bhutan, Sikkim and the

Kingdom of Nepal. It is likely that the varieties may have been originally imported from China for cultivation. Sporadic attempts to cultivate soya bean in other parts of India have been made but as yet the bean does not seem to have assumed the status of a food crop in India. One may make a conjecture about the underlying reasons. Firstly, soya bean or its products are not such as to permit a liking to be easily developed among people unaccustomed to them, mainly on account of their bitter taste and strong flavour. Secondly, India is rich in a variety of pulses and legumes, several of which have taken a firm foothold in Indian dietaries. Thirdly, it is quite probable (and for this the agricultural expert opinion is necessary) that climatic and soil conditions in many parts of India were not suitable for soya bean cultivation. The high protein and fat content of soya bean are considerations which are of nutritive and commercial importance. The United States of America was quick to see the possibilities and its cultivation in that country increased by leaps and bounds till in 1941 over 2.6 million tons were produced. Most of the crop is used for extraction of oil which is of a good edible variety. In the U. S. A. a great deal of work has been done on the nutritive value of the residual flour and of the proteins contained in it.

Several nutrition workers have at one time or other considered the soya bean as a panacea for the ills suffered by the poor people of the tropics allegedly on account of the low protein content of the diet. The high protein content of soya bean, its high yielding capacity and probably its cheap cost were the main arguments advanced by the exponents of soya bean. On the other hand, there was another school of thought in India which was of the opinion that

although soya bean had a larger protein content than several pulses, some of the latter were more nutritious on account of the higher biological value and digestibility of their proteins. They also argued further that the pulses were familiar articles of diet and it would be advisable to advocate their increased production and consumption, both of which were inadequate. Whereas the protagonists of soya bean derive strength from the work done in China, Far East and in the U.S.A., the opposing school based their arguments on actual experimental work in India, the results of which were already available. Such work was done mainly at the Nutrition Research Laboratories, Coonoor, South India, and at one or two other places. Taking all these facts into consideration, the Nutrition Advisory Committee of the Indian Research Fund Association had made the following recommendation at its third meeting held in New Delhi on 29th November 1937.

“**Soya bean**—The nutritive value of soya bean has been studied by experiments on animals, and also by controlled experiments on school children. The general conclusion is that soya bean, considered as a supplement to typical Indian diets, is not of outstanding value; it does not appear to have any advantage over various common pulses which have long formed part of the diet of the Indian people. While it would be advisable that results obtained in Coonoor Laboratories should be confirmed elsewhere, existing data suggested that at present the encouragement of the production and consumption of soya bean need not be made a prominent part of nutritional and agricultural policy in India.”

The difference of opinion between the two schools of thought continued to exist and came to a head in

1941 when the Nutrition Advisory Committee thought it best to reach an agreed conclusion in the interests of the problem itself and its likely effect on the policy with regard to soya bean cultivation in India. A small Sub-Committee was formed consisting of the warring elements and it was decided to undertake extensive investigation on soya bean in four different laboratories in India according to an agreed co-ordinated plan. The investigations commenced in 1942 and concluded in 1945. They embraced such questions as (a) biological value of soya bean proteins and other pulses by metabolism and growth methods, (b) the supplementary value of soya bean and pulses to poor rice diets, (c) the vitamin content and (d) the effect of soya bean and pulse supplements on the growth of school children. The results of the above work have been incorporated in the Sub-Committee's report published by the Indian Research Fund Association as Special Report I.R.F.A. No. 13, in January 1946. There resulted an almost complete agreement on the various points and the final paragraph of the report will illustrate how the recommendation more or less confirmed the view expressed in November 1937. The paragraph runs as follows:

“The Sub-Committee is not in a position, therefore to advocate *immediately* the encouragement of the production of soya bean on a wide scale in India for use as a substitute for Indian pulses. The question should, however, be reconsidered if and when further evidence on the nutritive value of soya bean becomes available.”

Just when the above co-ordinated investigations were about to conclude De and Subrahmanyam (1945) came out with a claim that by preparing a water



emulsion from germinated and then decorticated soya bean, a product was obtained which was palatable and in its nutritive value approached cow's milk. Their original method consists in a preliminary extraction of soya bean for 30 minutes at 70°C with an aqueous solution containing 0.5 per cent  $\text{NaHCO}_3$  and 1 per cent glycerol. The bean is then ground and mixed with four times its weight of water, boiled and strained through cloth. Sucrose, 2 per cent, and calcium lactate, 0.5 per cent, are added to improve taste and calcium content. Subrahmanyam and his colleagues have been busy during the years 1945-49 in improving their method of preparation and testing the product for its effects on the nutrition of rats and human beings. They found that the biological value of soya bean milk approached 90 per cent of that of casein and about 80 per cent of milk proteins. Desikachar and Subrahmanyam (1949) reported that in children of 1-4 years the calcium and phosphorus utilisation was identical with that of cow's milk although the utilisation of nitrogen of soya bean milk was lower. An experiment was conducted in orphanages on about 400 children where it was found that soya bean milk gave a growth response approximately equivalent to 80 per cent of that obtained with cow's milk. There is general agreement that soya bean milk can be a valuable supplement to poor rice diets although the nutrition workers in India still do not entirely agree with Subrahmanyam's interpretations regarding the comparative nutritive values of soya bean milk and cow's milk. Subrahmanyam himself is partly responsible for this: the method of preparing soya bean milk has been so altered on several occasions that it bears no relation to that first published by him. Unless one cares to sift carefully the evidence



presented by Subrahmanyan in a series of voluminous reports, it becomes confusing to interpret the results obtained with soya milk prepared by half-a-dozen different methods, none of which have been compared among themselves. Secondly, certain technical defects in the planning and conduct of human experiments render his conclusions open to question.

Subrahmanyan has attempted to make a case for soya bean by writing numerous articles on the subject, but for lack of support from agricultural experts on the one hand and nutrition workers on the other the position with regard to soya bean and our policy regarding its cultivation remains just where it was about 15 years ago when the Nutrition Advisory Committee made its first recommendation.

This somewhat lengthy account of the work and controversy on soya bean was considered necessary in order to provide the appropriate background for the scientific investigations and the attitude taken by a majority of Indian nutrition workers. In spite of the encouraging results obtained by Subrahmanyan on soya bean milk, the author still continues to be sceptic about the part which soya bean can play in improving the nutrition of the Indian people. At various times and from various directions quite irrelevant points have been put forth about the multiple uses to which soya bean can be put. The main point, however, must not be lost sight of. Soya bean is not an indigenous crop. We have several pulses which have all the advantages soya bean is supposed to have. Subrahmanyan's method of milk production is highly impracticable unless it is done only in large-scale plants. In order to cultivate soya bean in India, the land already under food crops will have to be weaned from the usual crops to be put under soya bean.

Finally, since people in general dislike soya bean and its products, its offtake and consumption will be inadequate. Therefore as a short term measure soya bean is no solution to our present nutrition problem. Even as a long term measure its success is doubtful and the authorities concerned will not embark on its large-scale cultivation unless they are assured by nutrition workers that soya bean possesses distinct advantages over the local foodstuffs. On this last point the expert opinion is definitely unfavourable with the sole exception of Subrahmanyam and his co-workers.

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## CHAPTER VI

### MILK AND MILK PRODUCTS

**Production:** The estimated production of milk in undivided India in the year 1940 was 48,080 million pounds and the total population then estimated amounted to 350 million. Another recent estimate (1947) placed the figure for milk production at 50,320 million pounds for a population of approximately 375 million people. The production, when calculated on a per capita basis, works out to have been about 5.6 ounces per day in 1940 and about 5.9 ounces per day in 1947. These figures may probably suggest a slight improvement in milk production. Too much significance, however, should not be attached to the small difference. It is safer to assume that milk production has remained in the neighbourhood of 5.6—5.9 ounces per capita per day. This rate of production is inordinately low and viewed in the light of the cattle population of India the yields are fantastically low. India has the largest cattle population in the world and also has the lowest milk yield. In 1940, there were 33.88 million cows and 12.64 million she-buffaloes; the yield for that year works out at 2.8 pounds per animal per day. For the year 1949 the corresponding figures for milch cows and buffaloes are 41 million and 19.6 million respectively. There has been, however, little improvement in the yield of milk per animal. The reasons for the low production of milk lie probably in (a) scarcity of fodder and concentrates, (b) general poverty of agriculturists and small producers and (c) lack of organised cattle breeding and dairy industry. The position

seems to have further deteriorated subsequent to partition of India. Sind and West Punjab were the two important milk producing centres. They were also the provinces from which good quality milch cattle was distributed to the rest of the country. With their incorporation in Pakistan, India has lost an appreciable part of good quality stock and the capacity for milk production has been proportionately reduced. It will take some time before the dairy industry in India can make up this loss.

The figures for total milk production do not give any idea of the consumption of milk as milk. In 1940, about 43.4 per cent of total milk produced was used for ghee (melted butter) manufacture, and in 1947, 55.6 per cent was used for making ghee. Recently, however, there has been reported a fall to the 1940 figure. Apart from this diversion of milk, there are other products prepared from milk which may account for an appreciable portion of the remainder, although it would be difficult to give exact figures. The official estimate for 1940 of fluid milk available for consumption as such was 1.64 oz./capita/day which means that 72 per cent of the total milk produced in a year, was being used for manufacture of milk products. Recent estimates give a slightly lower figure of 64 per cent for the proportion of milk used in the manufacture of all milk products.

The position is not so bad as these figures would indicate. The essential constituents of milk are retained in most of the milk products prepared for human consumption. In some the bye-products are also used as food. A short description given below will show the nature of these products and their nutritive value.

**Ghee:** Ghee is by definition butter fat from which water is removed by heating. The process of



making butter for ghee varies in details from the method used in the preparation of creamery butter. Fresh milk is brought to boil and allowed to remain at that temperature for five minutes or longer. It is then allowed to cool slowly and when warm to the touch inoculated with preformed curd. The lactic fermentation which proceeds may cause a firm curd to be formed between 8 to 36 hours depending upon the environmental temperature. An equal quantity of water is added to the curd and the mixture is churned in earthen or metallic vessels by a wooden churner. In 20 to 30 minutes butter separates which is then removed and pressed to remove excess butter milk. The practice varies from place to place; in some regions cream separated by slow heating of milk is removed, fermented and then butter is separated, in others milk is churned directly after cooling. In Indian homes the following practice is prevalent. Boiled milk is kept on a low heat when the cream gradually accumulates on the surface. This is carefully skimmed off and inoculated with preformed curd, covered and allowed to remain in a warm place. The lactic fermentation sets in and within 24 to 36 hours the mixture is ready for churning. Two to three volumes of water are added and the fermented cream is churned with wooden churn in an earthenware or metal pot. Within 20-30 minutes butter separates on the surface; this is collected and pressed lightly to remove excess water. The following table (Table X) gives the composition and certain analytical constants for Indian butter prepared according to the above methods; for comparison the figures for Indian creamery butter are also given in column three.

TABLE X—ANALYSIS OF INDIAN BUTTER

Particulars	Butter Indian Process	Butter Creamery Process
Moisture	8.5 to over 30 %	8.5 to over 16 %
Salt	nil	0.5 to over 2 %
Fat	70 to 85 %	75 to 85 %
Solids-not-fat	1 to 2.5 %	1 to 2.5 %
Casein	0.5 to 2 %	0.5 to 2 %
Free fatty acid	1 to 3 %	1 to 3 %
R. M. Value	24 to 28	24 to 28

The butter made from curd has a peculiar taste and flavour and Indians no doubt like them both. The butter made from cow milk is yellowish or orange in colour whereas that from buffalo milk is white. This difference is due to the fact that carotenoids are not present in buffalo milk.

The producer may himself convert butter into ghee or sell it to the consumer to be converted by the latter. The practice varies in different parts of the country. Butter is placed in a pan on the open fire and heated, the fat melts and when sufficiently heated the water boils and evaporates. The heating is continued on low fire till all the water has been driven off. The temperature of heating is necessarily over 100° particularly in the later stages of boiling and the time varies according to the quantity of butter being heated. The water-free product is known as ghee. It is cooled and usually sold in open containers or sealed tins. Ghee sold by small producers contains a little moisture and suspended impurities.

These are further removed in refining process in which ghee is remelted and held at temperature of about 70°C which causes settling of the suspended impurities and partial reduction of moisture content. The final product has a characteristic flavour and aroma imparted to it due to fermentation of milk or cream.

The above description will make it clear that there exists no standardized procedure for making ghee and hence there is little chance of obtaining a product which will retain the full nutritive value of butter fat. While it is true that the composition of fat does not change, the vitamin A content of ghee samples shows very large variations. It is probable that some vitamin A may be destroyed through the initial clarification of butter although Ahmad, Ramchand and Mansoor-ul-Hassan (1946) showed that heating butter at 150°C for 10 minutes caused only inappreciable vitamin A loss. These authors carried out a spectrophotometric assay on various samples and found genuine ghee (prepared from pure milk) to contain vitamin A from 14.6 to 40 I.U./gm. whereas market samples contained 16.6 to 24.6 I.U./gm. On storage at room temperature they found a loss of 30 per cent in 175 days. De, Ranganathan and Sundararajan found 8 to 11 I.U. vitamin A/gm. in some market samples from Madras, whereas for genuine ghee prepared from pure milk an average value of 36.5 I.U./gm. was obtained. Muthanna and Seshan (1941) have reported that ten samples from Sind contained 5 to 17.5  $\mu$ g./gm. and 9 Bengal samples 5 to 12  $\mu$ g./gm. of vitamin A. The vitamin D content of Indian butter and ghee has been determined by Dikshit and Ranganathan (1950) and values between 6 to 18.5 I.U./gm. have been obtained. These are

distinctly higher than the values reported by Coward and Morgan (1935) which are the ones generally accepted. How much the liberal exposure of the cow to tropical sun has to do with the high values of Vitamin D reported for Indian butter and ghee is a question which should require further investigation.

Ghee is consumed as such or may be used for cooking. In the former case ghee is usually melted and smeared on *chappaties* or mixed with *dahl*. The rice eaters add a small quantity to the helping of rice which is then eaten with curried *dahl* and vegetables. When ghee is used for cooking, it forms an ingredient of several sweet and savoury preparations. The other common use of ghee is for deep frying of *puries*. The temperature of frying is destructive for vitamin A and neither the ghee which is being continuously heated between 200 to 240°C, nor the *puries* fried in it retain any vitamin A originally present in ghee. Recently the use of ghee for these purposes has been on a decrease owing to its enhanced cost. It is being slowly supplanted by partially hydrogenated edible oils.

**Butter Milk:** It will be remembered that the by-product of butter prepared for ghee manufacture is butter-milk. This product is extremely palatable, and is very much liked by people who freely partake of it. Butter milk contains most other nutrients of milk except its fat. It can be taken as a beverage or can be eaten mixed with rice. The latter practice is common among the inhabitants of the peninsula whereas the northerners prefer it as a beverage. Butter milk is also a cheap substitute for curd.

**Curd:** The procedure of obtaining curd has already been described in the section on ghee preparation. Curd is a delicious food equally nutritious as

milk. Further, it has the advantage that persons who cannot tolerate milk can with impunity consume curd without any digestive disturbance. The inoculum for preparing curd is preformed curd or butter milk prepared from it. There are no precautions taken to retain the constancy of the microflora responsible for fermentation. This factor together with variation in time and temperature causes an appreciable variation in the quality, taste and flavour of the resulting curd. Pasricha and Goyal (1938) found *Lactobacillus acidophilus* in all 24 samples which they examined in Calcutta, and *Streptococcus lactis* in 23 out of 24 samples. They also found yeast in some samples. Joshi (1949) has identified and characterized a yeast in curd which he has named *Torula dahi*. Sundararajan (1950) mentions the presence of several other organisms in the microflora of curd. Ghosh and Guha (1935) found that curd had the same ascorbic acid content (0.7 to 1.0 mg. per 100 gm.) as milk from which it was prepared. Chitre and Patwardhan (1945) found that thiamine in milk was unaffected by curd formation, riboflavin showed a slight increase, whereas there was a marked decrease in the nicotinic acid content. In 16 milk samples they obtained values for nicotinic acid between 0.59 to 2.37 mg./100 gm. and in curds prepared from these same samples 0.25 to 1.0 mg./100 gm. nicotinic acid was obtained. It appears, therefore, that during fermentation some of the B vitamins are affected by the growth and metabolism of the microorganisms. Sundararajan (1950) could observe no significant change in the carotene and vitamin A content of milk on lactic fermentation. Desikachar and Subrahmanyan (1948) reported a better retention of calcium on cow or buffalo milk curd in six adult human subjects



than on an equivalent quantity of milk. This finding could not be substantiated in albino rats by Sundararajan (1950) who also showed that the biological value of milk and curd proteins by balance sheet or growth method as well as the supplementary value of these two food-stuffs to poor rice diets were identical. This confirms in some respects the earlier finding of Datta and Banerjee (1934) that the growth promoting capacity of curd was equivalent to that expected from a comparable quantity of milk. There seems to be a popular belief, however, that curd is more nutritious than milk, a belief which has not yet been substantiated by scientific investigations.

The other important milk products made from milk are *khoa*, *rabadi*, *malai* and *channa*. *Malai* is the cream which rises to the top on slow heating of milk; it is skimmed off and sold as such. *Rabadi* is whole milk boiled down to a thick consistency and sweetened. *Khoa* is whole milk concentrated in open pans till a granular solid still containing some water is obtained. *Khoa* forms a major ingredient of several Indian sweets. *Channa* is made by curdling milk either by rennet or by adding mineral or organic acids. The curdled mass is freed from whey, pressed and is made into sweets. It is true that several of these products are known all over India, still, as is only natural, they are produced and consumed in large quantities in the provinces of India where milk production is high, thus *khoa*, *rabadi*, *malai* are produced in large quantities in Punjab and United Provinces. *Channa* is the peculiarity of Bengal although some of it is prepared in Bihar and Orissa also. However, it must be mentioned that these products are sold at a comparatively high price and hence it is doubtful whether they would play any significant role in the daily diet

of the poor people of this country. Sweets prepared from milk are a luxury in any country and ordinarily out of reach of the common man except on certain occasions which must be considered to be rather rare.

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## CHAPTER VII

### PROTEINS

THE INVESTIGATIONS on the biological value (B. V.) of dietary proteins have been referred to earlier in more than one chapter dealing with the common Indian food-stuffs. It is no exaggeration to say that most workers interested in proteins have focussed their attention on this aspect in preference to others. Balance sheet and growth methods as applied to albino rats have been the ones most extensively used. Basu and De (1938) have determined the biological value of two Bengal fresh water fish *Hilsa* (*Clupea ilisa*) and *Ruhee* (*Labio rohita*) and found values of 70 and 79 respectively. Mitra and Mittra (1945) report values of 69, 60 and 60 for cow, buffalo and goat muscle meat respectively. These same authors (1943) have determined the biological value of proteins from red ants for which they report a value of 58.3 (at 10 per cent protein intake) with a digestibility coefficient of 65. It seems that a tribe of aborigines called Ho, inhabitants of Singhbhum District in Bihar, consider red ants a delicacy.

Mitra and Verma (1947) have investigated the biological value of proteins of mixtures of rice, pulse and milk on human subjects. They find that the B.V. of proteins of rice and pulse mixtures varying in proportions of 100 : 75, 100 : 100 and 100 : 125 ranged between 56.4 and 59.2, the differences not being significant. When milk replaced the pulse in the diet the B. V. rose to between 65.5 and 67.8. Mitra, Verma and Ahmed (1948) also determined in six human subjects the B.V. of proteins in cereal mixtures

with a nitrogen intake of 10.15 to 15.86 gm. daily. They found that with rice as the only cereal supplying protein, the B. V. was 66.6 whereas when rice was partly replaced with wheat, barley, maize, jowar, bajra, or ragi, singly or in mixture, the B. V. showed a decrease. The values for different mixtures ranged between 54.1 and 60.2. The digestibility of proteins on the other hand varied within the narrow range of 79 to 82 per cent. Mitra and Verma, therefore, feel that so far as the quality of protein is concerned nothing is gained by partial replacement of rice by other cereals.

Newer methods of evaluating the biological value of protein have recently been attempted by Subrahmanyan and his colleagues. Sahasrabudhe, Desikachar and Subrahmanyan (1947) studied the effect of casein and soya milk protein on regeneration of liver proteins and found that the latter gave a response equivalent to 81 per cent of that obtained with casein. Desikachar, De and Subrahmanyan (1948) used the same proteins in studies on regeneration of haemoglobin in rats rendered anaemic by phenylhydrazine injections and found that soya milk protein gave a response equivalent to 82 per cent of that obtained with casein.

The subject of protein metabolism in Indians is interesting. There appears to be a common impression, which, in the main is unsupported by authentic observations, that the diets of Indians in general are deficient in protein both qualitatively and quantitatively. The investigations on the biological value of cereals and pulses given elsewhere should convince anyone that although these proteins are somewhat inferior to meat, egg and milk proteins in their nutritive value, the difference is hardly such as would

justify the prevalence of such an impression. The question of the quantitative aspect of protein in diets will be discussed in a later chapter. At the moment we shall describe and examine critically the published evidence bearing on the metabolic aspect, which has given the erroneous impression referred to above.

McCay (1908) made about 200 observations on Bengali students, durwans (watchmen), household servants and *domes* and *mehetars* (depressed classes), in all 44 subjects, and he was greatly struck by the low levels of urinary nitrogen in all of them excepting one subject. The average urine nitrogen gave a value of 5.98 grammes daily. He argued that urine nitrogen indicated the level of protein metabolism in the body and in the case of normal healthy adults who were maintaining constant weights it gave an idea of protein intake as well. Whereas the first assumption is correct the second is not necessarily so as can be shown by reference to more recent work on the subject. McCay also mentions the fact that a majority of subjects had a perfectly free choice of food and were economically in a condition to take adequate diets. In view of this he considers that the only conclusion which his studies point to, is that Bengalis live on a diet the protein content of which is insufficient even by the Chittenden standards.

In the metabolism studies on two subjects, McCay found that on daily intakes of 8.635 and 10.56 gm. of N the amounts excreted in urine varied between 78-80 per cent; about 19 per cent appeared in faeces which he considered as unabsorbed nitrogen. In a further observation on 8 prisoners in a Calcutta jail he found on an average about 25 per cent nitrogen unabsorbed. From these results he concludes that in addition to the low intakes of protein, the low



digestibility of the latter in Indian diets was a contributory factor in the low level of protein metabolism of Indians.

The observations of McCay thus became the starting point for the assumptions regarding quantitative inadequacy and qualitative inferiority of proteins in Indian dietaries. Later workers appear to have uncritically adhered to these views. Thus Ray and Ganguly (1938) postulate merely on the strength of urine N determinations that since the average total urinary nitrogen was of the order of 4.83 gm. per day (for 50 individuals) their subjects must be ingesting about 35 gm. of proteins a day. Sokhey and Malandkar (1939) also assume a low habitual protein intake by Indians. Gokhale (1941) in Bombay observed in 47 male healthy adults an average of 6.09 gm. N per day with a range of 3.62 to 9.84 gm. In South India too, certain observations made at Madras and Coonoor have given similar figures. There is little doubt, therefore, that as a rule the average urinary nitrogen excretion of Indian adults would be in the neighbourhood of 5 - 6 gm. per day. If one assumes that in the faeces only 10 per cent of the ingested nitrogen appears, one is driven to the conclusion that Indians must be living on low protein varying between 35 to 40 gm. These assumptions themselves are open to question for the following reasons, (1) Indian adults are in positive N balance even at this low level of urine nitrogen, (2) the dermal losses, which are so important in a tropical humid and warm climate, have been completely ignored and finally (3) the influence of dietary protein on urinary nitrogen has not been taken into account at all.

Large positive nitrogen balances are known to occur during periods of active growth or during

convalescence after debilitating illness, but the fact that appreciable quantities of nitrogen can be retained daily by adults who have been maintaining constant weight has not been sufficiently well recognised. Grindley (1912) observed in 23 young adults, studied over a period of 220 days, an average nitrogen balance of 1.38 gm. per day per person. Commenting on these results, Mitchell (1949) states that even if one allows about 0.4 gm. N lost through insensible perspiration, a balance of 1 gm. nitrogen appeared to be retained per day. This can only be accounted for by assuming that it is utilized in replacing the amount lost through desquamation of cornified epithelial surfaces, sebaceous secretions, growth of keratinous material like hair, nails, etc. Apart from this, the capacity to store nitrogen compounds in readily available form cannot entirely be ignored. Kosterlitz (1944, 1947) has shown that fasting of even 24 to 48 hours' duration is accompanied by a loss of liver protein. Similar results are obtained, according to him, when rats are maintained on low protein diets or on proteins deficient in essential amino acids. Kosterlitz and Campbell (1945) suggest that a readily available mobile protein reserve is obtainable from liver cell cytoplasm and muscle. The mobile reserve may be augmented or depleted according to dietary conditions. This may, therefore, represent a part of nitrogen storage mechanism. The capacity of the subcutaneous tissue to store at least temporarily constituents removed from circulating plasma may be another factor about which not much is known.

The loss of nitrogen through sweat is an important consideration in nitrogen balance studies in the tropics. McCay's observations were made in Calcutta which has a warm climate with high humidity for

most months in the year. Although he mentions the possibility of such losses he does not seem to have realised the extent to which they would influence the interpretation of his results. Mitchell and Hamilton (unpublished) found that about 0.4 gm. N could be lost per day through insensible perspiration and Bricker, Mitchell and Kinsman (1945) place the probable dermal loss under excessive sweating at 4 gm. per day. The values for nitrogen lost through skin by McCay's subjects in Calcutta must have been nearer 4 than 0.4 gm. per day.

At the time McCay carried out his experiments there was no evidence to show that the type of dietary protein mixture could influence urinary nitrogen apart from the effect due to differences in the amounts absorbed. It was generally held that whatever protein was digested and absorbed from the gut and made available for metabolism should be reflected in the nitrogen of urine. This does not include the effects of such incomplete proteins as gelatin and zein which would cause body N losses to be evident in urine N. The work of Murlin and his associates was done at levels of N intakes which in minimum amounts maintained a person in equilibrium or in a slightly positive balance. In these experiments vegetable proteins caused a higher amount of N to appear in urine than egg protein. Now evidence will be presented to show that such differences in urine nitrogen occur at higher levels of N intake as well and in an opposite direction to that observed by Murlin. Data presented in Table XI illustrate how the shifts in urinary nitrogen take place with changes in the composition of diets chiefly with regard to protein.

TABLE XI—RESULTS OF NITROGEN METABOLISM IN INDIAN ADULTS

## PROTEINS

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Subject	Diet	Protein		Total N intake gm.	N in urine gm.	N in faeces gm.	Total N in excreta gm.	N Balance gm.
		Total gm.	Animal %					
K.M.	Basal	79.2	4.7	12.67	5.49	2.57	8.06	4.61
"	Experimental II	76.4	30.0	12.23	6.43	2.92	9.35	2.88
"	Experimental III	76.7	54.9	12.27	8.34	2.39	10.73	1.54
S.	Basal	76.3	4.9	12.20	4.45	2.35	6.80	5.40
"	Experimental II	78.4	31.7	12.54	6.39	2.70	9.09	3.45
"	Experimental III	81.8	58.2	13.08	8.43	2.82	11.25	1.84
B.V.R.	Basal	63.1	15.7	10.09	5.57	1.59	7.16	2.93
"	Experimental II	61.7	56.8	9.87	6.87	1.90	8.77	1.10
P.G.T.	Basal	61.4	16.1	9.83	6.61	1.31	7.92	1.91
"	Experimental II	60.1	62.7	9.62	8.46	0.90	9.36	0.26
P.V.K.	Basal	74.3	8.0	11.89	7.63	3.07	10.70	1.19
"	Experimental II	74.6	46.1	11.93	8.93	3.01	11.94	0.01
N.G.N.	Basal	56.9	11.6	9.10	4.95	1.38	6.33	2.67
"	Experimental II	53.1	49.0	8.50	6.53	1.37	7.90	0.60
G.K.W.	Basal	66.1	7.2	10.57	5.02	2.92	7.94	2.63
"	Experimental II	66.9	47.9	10.71	6.29	3.01	9.30	1.41

Pat.  
ward-  
han  
*et al*  
(1949)

Kar-  
ambel-  
kar  
*et al*  
(1950)

The experiments, results of which are given in Table XI, were carried out with the usual precautions taken in metabolic studies. The dietary pattern during the basal period was practically the same to which the subjects were habitually accustomed. Thus neither the calorie content nor the protein content was appreciably altered. The subjects were allowed to reach a steady state on the basal diet with respect to the urinary N excretion. Then faeces and urine were both collected for two consecutive days at a time with a gap of one day in between collections. When the results of atleast two successive periods were concordant the subjects were placed on a diet in which a certain percentage of protein was derived from animal sources; the total protein content remained unchanged or varied within very narrow limits. The subject was allowed to reach the steady state on this diet before urine and faeces were analysed for nitrogen. The diets were so composed as to permit the minimum alterations in the calorie value. The main alteration in the composition, therefore, was with respect to the type of the protein mixture. The results clearly show that on diets with predominantly vegetable protein, urinary nitrogen is lower than when approximately 50 per cent of total protein consists of animal protein. Identical animal protein effect is obtained whether the source is meat, milk or eggs. It is to be noted that all the subjects were either in a positive balance or reached equilibrium on certain diets.

It is noteworthy that similar results in all seven subjects have been obtained. That the degree of the effect depended upon the animal protein content is well brought out in subjects K.M. and S. These subjects when put back on basal diets reverted to the



original level of urine nitrogen excretion. Records of four other subjects have not been included but they also showed similar trends. It should be noted that although the faecal nitrogen varies from subject to subject there is no great variation on different diets in the same subject and hence the variations in the urinary nitrogen cannot be ascribed to differences in the digestibility of basal and experimental diets containing 5 to 63 per cent animal proteins. The protein in the basal diet was derived from a variety of cereals and pulses mainly and in animal protein periods different subjects were given meat, milk or eggs, the latter two singly or in mixtures. That this peculiar effect is thus brought about by a change in the type of dietary protein mixture and has nothing to do with digestibility is clear. In these observations and the predominantly vegetarian composition of Indian dietaries probably lies the secret of the low urine nitrogen observed in Indians and not in the low intake of protein or low digestibility of the protein contained in these diets. The cause(s) underlying the effect described above, however, still remain unexplained.

Another fact to which attention must be drawn, is the comparatively large nitrogen balance met with on vegetarian diets. These balances were continuous during the whole of the experimental period, yet weight changes among the experimental subjects were negligible, the subjects being either stationary or showing a small increase of two pounds over a period of two to three months. The question is whether such balances are a continuous feature in ordinary life or a greater part of N must be eliminated through the skin and possibly through other channels. Losses in sweat should be considered a strong probability

owing to the prevalence of tropical climatic conditions in India which must maintain an increased rate of perspiration throughout a large part of the year. Such large positive nitrogen balances in young healthy adults have been observed by others, e.g. Wilson and Mookerjee (1935), Basu and Basak (1939), Mitra, Verma and Ahmed (1948) but these authors have made no comments on the subject.

The level of protein metabolism in India as judged by urinary nitrogen is low as compared with Europeans and Americans. From the results obtained by Denis and Borgstrom (1924), Beard (1927), Brooks (1929), Martin and Robison (1922), Johnson, Deuel, Morehouse and Mehl (1947), it appears that 147 to 200 mg. per kg. body weight is the range of urine nitrogen excretion. The Indian values [Basu and Basak (1939), Basu, Basak and De (1941), Niyogi, Patwardhan and Sirsat (1941), Karambelkar, Patwardhan and Sreenivasan (1950)] fall between 66 and 160 mg./kg. with an average of 114 mg./kg. If these figures are expressed per square metre of body surface those for Europeans and Americans average at 6.11 gm./N/m<sup>2</sup> and in Indians 4.3 gm./N/m<sup>2</sup>. These facts, however, do not necessarily lead to the conclusion that the level of total protein metabolism in Indians is low. Conclusions based on urine nitrogen only are not valid for in the tropics and subtropical regions the role of the skin in determining nitrogen loss should be quantitatively of greater importance than in the temperate regions.

**Endogenous Protein Metabolism:** There is little evidence to show that this differs from the accepted standards. Basu and Basak (1939) placed three subjects on protein free diets; Mitra, Verma and Ahmed (1948) placed six subjects on low protein

diets and Karambelkar *et al* (1950) observed three subjects on similar diets. The average values observed by all these investigators recorded at Dacca, Patna and Bombay respectively are given in Table XII.

Terroine (1936) mentions that the highest values recorded for 'endogenous nitrogen expenditure' barely reach 3 gm. of N per day for a man of 70 kg. body weight. When expressed per unit of body weight, Terroine's expectation will give a figure of approximately 43 mg. N per kg. which is similar to the last two figures in Table XII, Column 8.

The reported figures for urinary excretion of creatinine in Indians throw further light on the subject. It is true that the status of creatinine as an infallible index of endogenous protein metabolism has been in doubt since the researches of Schoenheimer. However, the constancy of creatinine excretion in urine from day to day could be considered to have at least some relation to the level of endogenous protein metabolism of the body. Niyogi, Patwardhan and Sirsat (1941) found a creatinine coefficient between 19 and 27 in 10 subjects and Karambelkar *et al* (1950) observed values between 19.2 to 23.4. The reported figures for Americans vary between 20 to 26. The observed total creatinine excretion in 17 Indian subjects varied from 1.0 to 1.7 gm. per day as against the reported American figures of 1.5 to 2 gm. The slightly lower excretion of creatinine in Indians could be ascribed to a smaller protoplasmic mass (including musculature) responsible for the low body weights. It should thus be clear from the above discussion that the endogenous protein metabolism appears to be on the same level as in Westerners and that the small differences which are found between the two groups

TABLE XII—ENDOGENOUS NITROGEN EXCRETION IN INDIAN ADULTS

Authors and Place	No. of Subjects	N Intake gm.	N in urine gm.	N in faeces gm.	N Total output gm.	N net output gm.	Endogenous N mg. / kg.
Basu <i>et al</i> (1939)							
Dacca	3	2.0	2.124	1.071	3.195	—	66.3
Mitra <i>et al</i> (1948)							
Patna	6	0.39	1.522	0.963	2.485	2.019	46.5
Karambelkar <i>et al</i> (1950)							
Bombay	3	0.46	1.67	0.95	2.62	2.16	41.4

can be reasonably explained by the differences in body weight.

In view of the above findings on endogenous metabolism it becomes difficult to find an explanation for differences in urine nitrogen on different dietary proteins, particularly as the observed differences in digestibility are too small to afford a satisfactory explanation. Positive balances of nitrogen may be only apparent on account of the dermal losses not being considered. It is the influence of dietary protein on urinary nitrogen, however that requires further careful investigation.

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## CHAPTER VIII

### FATS

VEGETABLE OILS are extensively used in India as cooking fats. The use of lard and suet, objected to by the two major communities on religious grounds, is limited to an extremely small—almost negligible—fraction of the Indian population. At one time ghee was being used for cooking purposes, particularly in North India, but on account of its increasing scarcity and high cost, its place has been taken by oils, chiefly the partially hydrogenated oils. The main varieties of vegetable oils used in India are groundnut oil, mustard and rapeseed oil. Sesame oil, cocoanut oil and safflower oil, linseed oil and mahua (*Bassia latifolia*) oil are also used in certain regions within the country. Bengal, Bihar and part of the United Provinces are partial to mustard oil, Bombay to safflower seed oil and a large part of the Deccan to groundnut oil. Cocoanut oil is used exclusively on the west coast and gingelly or sesame oil is favoured in South India. In the north, rapeseed oil and sesame oil are both used to a large extent. These oils are mainly used for preparing various spiced and curried foods and for frying during the course of the preparation of other foods.

The composition and characteristics of these edible oils have been studied by Sudborough and his school at the Indian Institute of Science, Bangalore. As the purely chemical findings are not of particular interest from our standpoint no further mention of that aspect is made in this book.

The hydrogenated oil products were first introduced in India in the third decade of this century.

They were mainly imported from Holland. In 1930 their manufacture was started in India. Since then the industry has made rapid advances as can be shown by the fact that whereas in 1935 only 18,000 tons of hydrogenated products were sold in India, in 1946 the amount sold was 137,000 tons. The imports after 1930 have been negligible on account of heavy import duty, and hence almost the whole of the product sold in the country can be considered to be of indigenous manufacture. Groundnut oil forms the chief raw material for the industry although rapeseed and sesame oils have also been recently used to a certain extent. The immense popularity of the hydrogenated oil is due to an improvement in stability and physical appearance of the fat as well as better cooking quality. The phenomenal growth of the industry during the short span of 15 years frightened the ghee manufacturers, for in it they saw the possibility of injury to ghee industry. Although these fears were groundless the fact that hydrogenated oils are also used for adulteration of ghee has been instrumental in turning the public opinion against the former. The unfortunate publicity given to certain incomplete investigations led the people to believe that hydrogenated oils were a positive danger to public health. At one time the public had genuine doubts about the wisdom of continuing to consume hydrogenated oils which were generally known by the names of 'vanaspati' and 'vegetable ghee'. The Government of India had to take notice of the popular anxiety in this respect and they appointed a Committee of scientific experts to investigate the comparative nutritive value of hydrogenated oils. This Committee was well aware of the work done in other countries which had shown that on adequate

diets the nutritive value of different edible fats was of the same order. But it was argued that as the bulk of the Indian population lived on marginal, if not inadequate, diets the differences in the nutritive value of fats, if there were any, would be of significance. Hence it was felt necessary to design and conduct experiments on rats and human beings to settle this point. Several laboratories collaborated in the co-ordinated investigation and after three years of work the Committee concluded that between the partially hydrogenated (m.p.  $37^{\circ}$ - $39^{\circ}$ ) and unhydrogenated vegetable fats there was no difference in the nutritive value, whether fed with adequate or inadequate diets. This opinion of the Committee has been conveyed to the Government of India and through them to the public and it is hoped that this will properly settle the controversy which really should never have arisen in the first instance. One good thing has come out of this, the interest of workers in fat studies has been aroused and the impetus given by the controversy has resulted in sustained efforts designed to study the role of fat in Indian diets.

Apart from the above, considerable work has been done on the comparative nutritive value of different edible oils. The results in some cases are contradictory, but there exists a large measure of agreement on certain important points. The absorption of fats has been studied in rats as well as in human beings. The digestibility coefficients as well as the rates of absorption have also received attention. Roy (1944) found that the digestibility of common edible oils and ghee varied between 95.5 and 99.7 per cent, thus showing no significant difference in the extent of absorption of ingested fats. Misra



and Patwardhan (1948) found in rats no differences in the digestibility of groundnut oil and its partially hydrogenated products up to a m.p. of 49 C. These authors also studied the digestibility of hydrogenated groundnut oil samples containing 19 and 29 per cent iso-oleic acids and could not detect differences between the two. In human beings digestibility trials gave results which again showed lack of significant differences between different fats [Basu and Nath (1946); Tulpule and Patwardhan (1948) and Subrahmanyam *et al* (1949)], the digestibility coefficients being above 90 per cent for all the fats investigated. In these investigations hydrogenated groundnut oil of m.p. 37° was also included. When a product melting at 41 C was used the digestibility was slightly and significantly lower than 90 per cent. Subrahmanyam (1949) observed that straight hardened vegetable oils and the blended product of comparable melting point gave similar values for digestibility.

The rates of absorption have been studied in rats by Basu and Nath (1946). 1 to 2 gm. fat was introduced in the stomach of adult rats by stomach tubes, the rats were killed at 2, 4 and 6 hourly intervals and the fat remaining in the gastrointestinal tract was determined. The differences noticed in the rates of absorption were slight and not significant. The work of Anantakrishnan (1945) at the Indian Dairy Research Institute, Bangalore, has also shown only minor differences at four hours between the amounts of various edible oils and ghee absorbed. There was one exception, however. Mustard oil was the least absorbed, it gave a value of 27.1 per cent absorption as compared to the values 38.9 to 48.4 per cent obtained with other edible oils and ghee made from milk of various animals. Basu and Nath had also

observed a slower rate of absorption for mustard oil at two hours, but their values for 4 and 6 hour periods are not much different from those obtained with other oils. Anantakrishnan found a lowering of the rate of absorption in rats when hydrogenated product was used in place of oil. Similar results were obtained with groundnut, sesame and cotton seed oils and their hydrogenated products of m.p.  $38^{\circ}$ - $39^{\circ}$ C.

Nhavi and Patwardhan (1946) utilised Frazer's chylomicrograph technique in the study of comparative rates of absorption from the human intestine. The test meal consisted of 2 oz. bread, 1 cup (8 oz.) of sweetened tea and 1 oz. of fat under investigation taken on an empty stomach. The maximum chylomicron count was reached between  $2\frac{1}{2}$  to 3 hours with butter or ghee. Ghee heated to  $240^{\circ}$ C for a few minutes for frying was absorbed at a rate only slightly slower than that of untreated ghee. The rate of absorption of cocoanut oil was similar to that of butter. On the other hand, groundnut oil, sesame oil and hydrogenated groundnut oil gave the maximum count between 5 to 6 hours (Figure IV).

From their observations the authors were led to suggest that the presence of lower fatty acids as components of the glyceride molecule was probably more effective in determining the rates of absorption than differences in iodine value. The observations of Nhavi and Patwardhan on butter and vegetable oils were confirmed by the work of Subrahmanyam *et al* (1949). These results, however, do not agree with those obtained with rats by a different technique.

The quality of edible fats offered for sale to the consumer is uncontrolled and often indifferent. A mention of the adulteration of ghee has been made

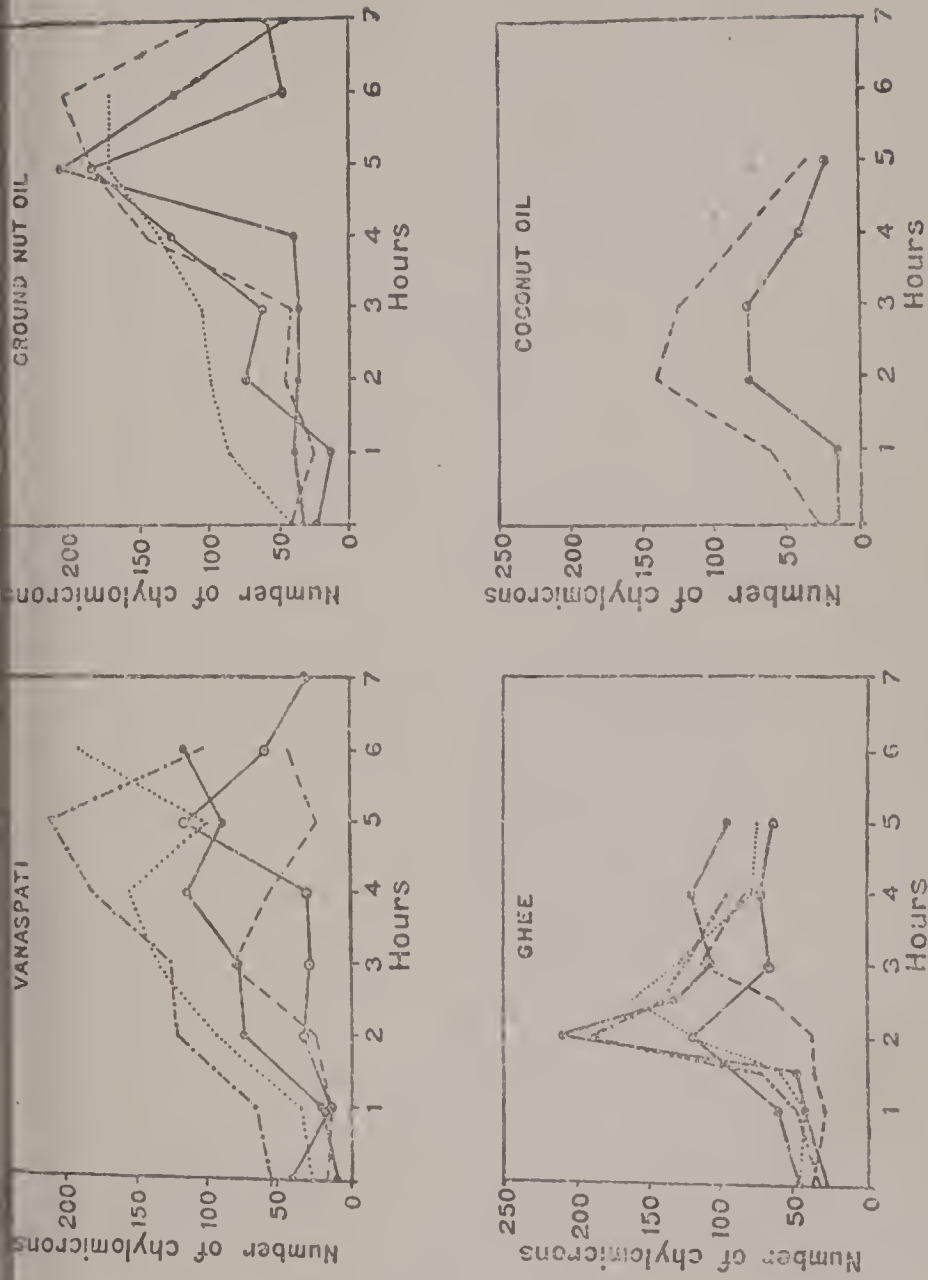


Figure IV—Rate of absorption of edible fats from the human intestine. Each curve represents one human subject. (By Courtesy of Indian Journal of Medical Research)

already. Apart from hydrogenated oils, other important adulterants in frequent use have been refined vegetable oils and even animal tallow. Adulteration is unchecked and probably has increased considerably in extent on account of the scarcity of ghee and the resultant high cost. The vegetable oils themselves are adulterated in actual practice. White oil is a favourite adulterant which several vendors use; this malpractice has increased to such an extent, that it has already become a menace to public health. Mustard oil, adulterated with argemone oil, wilfully or otherwise, has been responsible for numerous outbreaks of epidemic dropsy in Bengal. One is left with the strong impression that food laws are quite inadequate and business morality has reached a new low standard. It will be a herculean task to set matters right.

Apart from adulteration, the quality of the unadulterated product itself causes concern. Oils of varying acidity and peroxide values are on the market. These two conditions are unfavourable for the stability of dissolved vitamin A and carotene. As most vegetable oils do not contain carotene and none contains vitamin A, the problem would have been of academic interest were it certain that rancid oils exerted no influence on carotene and vitamin A introduced in the intestines with other foodstuffs. In any case the suggestion of Ramamurti and Banerjee (1948) that the sale of oils showing free acid beyond a certain limit should not be permitted for edible purposes is certainly worthy of consideration.

Some other investigations on fat may be mentioned in passing. Basu and Nath (1946) reported differences in growth promoting value of mustard, cocoanut, sesame and groundnut oils in rats. They found that

cow butter gave significantly better growth and even among oils there were significant differences. These conclusions have not been substantiated by later work, notably that of Anantakrishnan and co-workers. Basu and Nath used comparatively a small number of animals and their vitamin supplements about which they give no quantitative information were probably variable and inadequate since no pure vitamins were used. Mason (1944) has made an interesting observation in albino rats on the growth depressing action of butter when added to poor rice diet. Similar growth inhibition in rats was obtained when cocoanut, sesame or groundnut oil was added to a poor rice diet. Mason, Theophilus and Frimodt-Møller (1945) found that when casein was added with butter to the poor rice diet, growth rate was considerably accelerated, and with the addition of calcium lactate, growth was still further accelerated. Similar observations on the supplementary effect of butter and ragi were made by Mason, Devadas and Frimodt-Møller (1946). The depressant effect of addition of fat to poor rice diet has been observed by other workers also, but an adequate explanation is still lacking.

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## CHAPTER IX

### CALCIUM, PHOSPHORUS AND OTHER MINERALS

AMONG the mineral components of the diets the absorption and metabolism of calcium and phosphorus have received detailed attention at the hands of Indian workers. Chitre and Patwardhan (1940) studied the absorption of calcium from the intestines. Working with anæsthetized dogs they observed that on the introduction into the duodenum of calcium chloride or calcium lactate solutions the concentration of calcium in the portal blood and mesenteric lymph increased simultaneously. The preabsorption level of serum or lymph calcium, the amount of the salts introduced or the pH of the solution within the limits investigated had little influence on the rate or extent of rise of calcium in lymph and portal blood. The authors suggested that the absorption of calcium proceeds by the lymphatic as well as the portal venous routes. That both these routes are open for the transport of absorbed materials from the intestine and that only the (*a*) permeability of lymph and blood capillaries and (*b*) the rate of circulation are the dominant factors in determining the respective amounts transported by these two routes had been demonstrated by Bolton and Wright (1937) in their studies on digestion and absorption of peptone. These observations had been extended by Patwardhan and Nhavi (1939) to the study of absorption of phosphates. When phosphate solutions were introduced into the duodenum of anæsthetized dogs, changes in the total and inorganic phosphorus of

lymph and portal blood occurred which proved that the lymphatic and portal channels both were participating in the absorption. These authors also found that at pH 9.4 the absorption of inorganic phosphate was rapid whereas it decreased at pH 7.0 and was slowest at pH 4.9. Glycerophosphate was absorbed almost as rapidly as inorganic phosphate at pH 4.9 as was shown by the rise in the blood and lymph of total and inorganic P. Sodium phytate, however, at pH 3.8 or 5.2 was not absorbed. This was probably due to the fact that it was not hydrolysed in the intestine owing to the absence of enzyme phytase.

Patwardhan and Chitre (1942) studied the effect of vitamin D on the absorption of calcium from the intestine of the albino rat. They used Verzar's (1936) isolated loop technique in normal rats, rats rendered rachitic, and rats rendered hypervitaminotic by massive dosage with vitamin D. Calcium gluconate was introduced into the loops and the amount remaining after particular intervals determined in the loop washings. They found no significant differences in the amounts absorbed in the three groups of rats. Their observations, thus, did not confirm the report of Nicolaysen (1937) that in rickets the absorption of calcium from the intestine was defective. On the other hand, they concluded that vitamin D had no influence on the absorption of calcium from the intestine. These observations do not explain the well known fact that in rickets more calcium is lost in faeces and that on administration of vitamin D the faecal calcium decreases. Recently a suggestion has been made by Harrison and Harrison (1950) that the intestinal absorption of calcium is a summation of two phases, an initial rapid phase and a slow phase. The rapid phase, according to

these authors, is uninfluenced by vitamin D whereas in the slow phase calcium absorption is increased under the influence of vitamin D. If this be true the observations of Patwardhan and Chitre can be satisfactorily explained.

The availability of calcium and phosphorus from different foodstuffs has been determined by several workers. Ranganathan (1935) found in rats that retention of calcium when the latter was supplied by *bajra* (*Pennisetum typhoideum*) and *ragi* (*Eleusine coracana*) was less than 50 per cent whereas the calcium of whole wheat, *jowar* (*Sorghum vulgare*) and polished rice was retained to the extent of 70 to 87 per cent. The retentions of phosphorus from these different foodstuffs were, however, not markedly different. Giri (1940) observed that at lower levels of intake the utilisation of *ragi* calcium was higher. He also found that on cereal diets a major portion of faecal phosphorus in rats was in the form of ester phosphorus. Rau and Murthy (1942), Basu, Basak and De (1942) and Basu and Ghosh (1943 a) have determined the availability of calcium from green vegetables. The last named authors (1943 b) found in one human subject that calcium retention from cabbage, lady's finger and amaranth was lower than that from milk. These experiments require to be extended. Karnani, De and Subrahmanyam (1948) found the calcium from fortified soya bean milk well utilised by rats. Karnani *et al* (1948) studied the same problem in children 7 to 8 years in age. Using the method of Steggerda and Mitchell (1939) they found that calcium from soya bean milk was utilised almost to the same extent as that from cow milk.

The human requirements of calcium have also been studied although inadequately. Basu, Basak

and Rai Sircar (1939) report that for Indians 0.388 gms. of calcium and 1.00 g. of phosphorus may be considered as maintenance requirement. Nicholls and Nimalsuriya (1940) suggest that 0.5 gm. of calcium daily should be sufficient. The American and European standards are considered high by them, but Sherman's figures for requirements, shed of their safety margin appear close enough to the above figure. Further information on the requirements of calcium and phosphorus is obviously necessary particularly in view of the effects of certain dietary factors on their metabolism which have recently been investigated by Desikachar and Subrahmanyam (1948, 1949).

## IRON

The amount of total iron in Indian diets appears to be more than adequate, yet there are reasons to believe that the wide prevalence of microcytic anaemias is mainly due to iron deficiency. It is probable that only a part of the dietary iron is physiologically available. Roy, Pal and Guha (1939) determined the total and ionisable iron in the dietaries in eight students' hostels and found values of total iron between 34.6 to 39.7 mg. per head per day; only 18 to 25 per cent of this was ionisable iron. Lehmann (1949) has studied the 'available' iron in wheat dietaries of North-West India. He determined the total iron content of these diets by diet surveys and by analyses of foodstuffs and found it to be between 22 and 27 mg. per day. Only 39-40 per cent of the total iron was in the form of 'available' iron. Although there are no comparable data on dietaries elsewhere, the similarity of foodstuffs entering into the composition of Indian dietaries leads one to believe that 'available'



iron may be between 18 and 40 per cent, i.e., between the two extremes of the recorded observations. Ranganathan (1938) reports wide variations in the iron content of foodstuffs notably in green leafy vegetables and hence the calculation of iron content of diets may lead to wrong estimates of intake. He quotes the work of Hahn and Whipple (1938) on utilisation of iron and its salts and agrees with their suggestion that all the ionisable iron need not necessarily be considered as physiologically available. Saha and Guha (1940) observed that in certain animal tissues, e.g., fish muscle, a part of physiologically available iron exists in combination with protein; it can be liberated by peptic digestion and then can be estimated by the dipyridyl method of Hill. Pal (1939) determined the ionisable iron in human and cow milk and found that 98-99 per cent of the total iron in the former and over 90 per cent in the latter was ionisable. Saha and Banerjee (1943) have compared the absorption from isolated loops of rat intestines of ferric chloride, ferrous ammonium sulphate and Fe-Cu-nucleoprotein complex isolated by Saha (1941) from fish muscle. They found that iron in all the three was equally well absorbed.

Guha and his school have devoted considerable attention to the iron complexes of fish muscle and egg yolk. Iron in fish muscle according to them exists in a Fe-Cu-Protein complex and is resistant to peptic hydrolysis. This iron is, however, found available for hæmoglobin formation in anæmic rats. These authors have purified the Fe-lecithovitellin complex and used it for comparative hæmopoiesis studies. They find that iron from this complex when compared with  $\text{FeCl}_3$  is equally well utilised by anæmic rats for hæmoglobin regeneration, whereas iron

from wheat, rice and pulse (lentil) is poorly utilised. Phytin content of the latter probably plays a role in determining its availability in these foodstuffs. If the results obtained in these investigations are any indication they lend further support to the statement made at the beginning of this section, that in spite of adequate amounts of total iron in the diets there might still exist iron deficiency owing to the variable availability of the element. It is, therefore, imperative that we know more about the biological availability of iron in various foodstuffs.

There are two other mineral elements, iodine and fluorine, which will have to be considered in relation to nutrition and health in India; for both the iodine deficiency goitre and the toxic effect (fluorosis) of chronic excess of fluorine intake are conditions found in certain parts of India. As it is considered more appropriate to discuss work on iodine and fluorine while dealing with these diseases, they are not being dealt with in this chapter.

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## CHAPTER X

### CAROTENE AND VITAMIN A

THE IMPORTANCE of carotene in Indian diets as a source of vitamin A can hardly be overemphasized. As mentioned later in the chapter on Diets, the bulk of the Indian population ingests in negligible quantities foodstuffs containing preformed vitamin A. It is mostly the carotene in different vegetables and fruits which is utilised for conversion into vitamin A. It is, therefore, not surprising that the occurrence of carotene and related pigments in vegetables and foodstuffs has been studied in detail. Already we possess information on the total carotenoid content of over 200 common Indian foodstuffs. The vitamin A activity of different carotenoids varies greatly and hence the recent studies of Ahmad and his school on the identification of the carotenoids in plant foods is worthy of mention. These workers have examined tomatoes, mango, carrots and maize and have shown that true vitamin A activity can be calculated with reasonable accuracy if the carotenoid composition is known. Sadana and Ahmad (1946 a) identified in maize from Punjab, zeaxanthin, cryptoxanthin, neocryptoxanthin  $\alpha$ -carotene and  $\beta$ -carotene, the total pigments amounting to 18.8  $\mu$ g/gm. Taking into consideration the varying vitamin A activity of these pigments the authors gave for medium yellow maize a vitamin A value of 6.5 to 9.0 I.U./gm. and for dark yellow to deep red varieties 10 to 12.5 I.U./gm. Using similar technique Sadana and Ahmad (1946 b) give vitamin A values of 8–67 I.U./gm. for 10 different varieties of mango. Sadana (1947) has reported great

variations in vitamin A values of carrots, red varieties being the richest with 85 to 194 I.U./gm. and yellow the poorest with only 0.5 to 2.3 I.U./gm. Bahl, Sadana and Ahmad (1949) report that in tomatoes the concentrations of lycopene and  $\beta$ -carotene differ in different varieties, intervarietal crossing gives intermediate values for lycopene but  $\beta$ -carotene content is depressed. It must be admitted that investigations of this kind are important for till recently it has been the practice to lump together several carotenoids and calculate the vitamin A value by the use of arbitrarily chosen factors. It will be necessary to evaluate a large number of vegetable foodstuffs to obtain the correct vitamin A values based on their different carotenoid content.

De (1935, 1937 a and b) has done considerable work on the spectrophotometric assay of vitamin A and carotene. He took advantage of differential rates of destruction of vitamin A and carotene when exposed to ultra violet light in devising a simple method for estimation of the two constituents where both were present. Later he showed that from the ether or petroleum ether solutions of oils containing vitamin A and carotene the latter could be removed by adsorption on charcoal. In the presence of glycerides the adsorption of carotene was found to be almost complete, while that of vitamin A was negligible. Similar treatment of the nonsaponifiable fraction, however, made no difference in the adsorption of carotene although that of vitamin A was appreciably increased. The presence of traces of hydroquinone assisted in the separation.

De (1936) has determined the carotene content spectrophotometrically in vegetables, fruits and other Indian foodstuffs. Ahmad, Mullick and Majumdar



(1937) independently estimated carotene tintometrically in a large variety of foodstuffs. Most of these values will have to be interpreted with caution in the light of later work by Ahmad *et al.* It is necessary, as mentioned earlier, that extensive investigation to determine the true vitamin A activity of the important plant foods in India is undertaken to provide the requisite information.

A reference to the vitamin A content of ghee (clarified butter) and that of milk has already been made in the chapter dealing with milk and milk products. Another material which attracted a good deal of attention for its vitamin A content during the last ten years deserves mention. Fish liver oils, particularly those of saw-fish and Indian shark, were found by several workers in India [Seshan (1940), Majumdar (1941), Rajagopal (1941) and Niyogi *et al* (1943)] to contain appreciably large amounts of vitamin A. The product known as shark liver oil is now being manufactured in large quantities and at one time completely replaced cod liver oil in the Indian market when its import was either restricted or negligible during the war years. Most of the determinations of vitamin A content in fish liver oils were done either colorimetrically or spectrophotometrically. Rajagopal and Seshan compared the two methods using the whole oil and the nonsaponifiable fraction. Working with marine fish (shark and saw fish) liver oils Rajagopal found that the colour intensity with nonsaponifiable fraction was, on an average, 1.60 times that obtained with the equivalent quantity of the whole oil, a ratio which agrees well with that reported by Dyer (1933) on cod liver oil. Rajagopal agrees with the suggestion made by other workers that the differences may be due to the removal of "inhibitors" and/or the

hydrolysis of vitamin A ester during saponification of the oil. The average ratio of  $\frac{\text{International Units}}{\text{Blue Units}}$  varied very widely with the whole oil but with non-saponifiable fraction the variation was less. The author recommends the conversion factor of 53 which he finds to be in close agreement with factor 55 recommended by Seshan (*loc. cit.*). Both these workers have used 1600 as the conversion factor for expressing vitamin A content from the figure for  $E_{1\text{ cm}}^{1\%} 328 \text{ m}\mu$ .

Attempts to compare the biological activity with the calculated values from tintometry were made by Ghosh and Guha (1935) who found that the biological activity (in rats) of vitamin A preparations varied according to the solvent used for diluting the vitamin concentrates. On the whole, however, they found some agreement between tintometric and biological values. Datta and Banerjee (1934) also reported good agreement between the two methods of assay. The liver oils used by the two sets of authors originated from the same varieties of fresh water fish, e.g., *Labio rohita* and *Cirrhina mirgala*. Niyogi *et al* (*loc. cit.*) compared the vitamin A value of liver oils of two marine fish *Sciæna miles* and *Scoliodon sorrokowah* by tintometric and biological assay methods. The former method gave 7,300 I.U. and 42,000 I.U. vitamin A per gm. of oil respectively. The average results of two biological assays on each sample were 7,260 and 30,320 I.U./gm. respectively. The authors, therefore, cautioned against uncritical acceptance of either the tintometric or spectrophotometric results on new fish liver oils. Rao (1946) and more recently Bose and Subrahmanyam (1949) have studied the

conditions under which the vitamin A in fish liver oils is destroyed. The latter authors have confirmed the suggestions of Bose and Banerjee (1945) regarding the steps to be taken to protect the vitamin from such destruction. A combination of isobutyl gallate and tartaric acid or citric acid (0.02 % : 0.01 %) gives very good protection.

*Absorption and utilisation of carotene and Vitamin A:* De (1937) studied the absorption of carotene and vitamin A from the gastrointestinal tract of the albino rat. He found that 45 to 65 per cent of carotene was absorbed from red palm oil, orange juice, papaya and amaranth leaves. The body weight of the animal and the presence of fat, minerals or dried yeast did not materially influence the absorption of carotene. Under similar conditions vitamin A was almost completely absorbed. De and Majumdar (1938) could find no difference in the extent of absorption of carotene in weanling rats kept on poor South Indian diet as compared with rats of comparable age but previously depleted of vitamin A. Wilson, Ahmad and Majumdar (1936) had observed that the presence of fat in the diet assisted carotene absorption in vitamin A depleted rats. A similar observation has been recorded by Wilson, Das Gupta and Ahmad (1937) in an adult human subject. On a 10 to 30 mg. intake of carotene this subject absorbed 78 to 87 per cent of carotene when mustard oil and ghee were included in the diet. When the fats were omitted the absorption was only 52 per cent. On the other hand, 15,000 to 30,000 I.U. of vitamin A were almost completely absorbed irrespective of the presence or absence of added fat in the diet. Majumdar (1939) kept vitamin A depleted rats on nearly fat free (0.4 per cent fat) diets and administered to them

orally a colloidal suspension of carotene in 2 per cent glucose. From a study of the improvement in the clinical condition and in liver storage of Vitamin A, he concluded that fat was not necessary for the absorption of carotene. This may be so but as other experiments have shown the absorption may be greater when fat is also present.

Ahmad and Malik (1933) and Ahmad, Grewal and Malik (1934) have observed certain species differences in the capacity to utilise carotene. Rats, cats, rabbits, pigeons and chicks were first depleted of vitamin A and then fed 0.5 mg. carotene (from carrots) per kg. body weight per day for eight weeks. The authors found that rat had the highest storage of vitamin A in liver and cat none. They conclude that cat cannot convert carotene into vitamin A but it can store the latter if administered preformed. When colloidal aqueous suspensions of carotene were injected intraperitoneally or intramuscularly in rats, dogs, goats and rabbits, the authors could find conversion into vitamin A only in rabbits. Later, Wilson, Ahmad and Majumdar (1937) found an increase in vitamin A and decrease in carotene content of an autolysing liver from a rabbit given 5.4 mg. carotene intravenously. When carotene was, however, fed orally no such conversion could be observed, an experience which was similar to that observed in rat livers. The authors express a doubt whether the conversion of orally administered carotene normally would take place in the liver, which merely confirmed the unsatisfactory state of our knowledge on the subject. The nonutilisation of parenterally administered carotene was later confirmed by Sexton, Mehl and Deuel (1946), who showed that under the circumstances carotene merely accumulated in the liver.

On the other hand, the conversion of orally fed carotene to vitamin A had been repeatedly observed by various workers. Sexton *et al*, therefore, suggested that intestine was the site of conversion. In 1947, Mattson, Mehl and Deuel produced evidence to show that the wall of the intestine was the site of conversion. This has been confirmed by Glover, Goodwin and Morton (1947). Certain experiments carried out at Coonoor (1949) showed that while the wall of the intestine was involved in the conversion a certain amount of vitamin A was also found in the intestinal lumen of rats to whom carotene had been administered orally. Similar observations were reported by Thompson, Ganguly and Kon (1949). It appears, however, that the evidence for conversion in the lumen itself is still tenuous, considering the large doses of carotene given and the extremely small amounts of vitamin A found in the intestine at any time.

Hassan and Khanna (1947) and Hassan, Ibrahim and Khanna (1948) have determined the vitamin A blood levels in 108 men and 15 women medical students in Lahore. Their average values are as follows :

		No.	Per 100 c.c Plasma	
			Vitamin A I. U.	Vitamin A plus Carotene I U.
Men	...	48	109	202
Women	...	15	91	190
Men II Series	...	60	125	...



Hassan and Khanna used a modified Jeans-Zentmire apparatus for determining the dark adaptation of their subjects and found that the plasma levels lower than 60 I.U. were associated with impaired dark adaptation. Some other observers in India also have studied dark adaptation, but none has tried to correlate its impairment with blood levels of vitamin A [Rajagopal (1941), Basu and De (1941)]. Khan (1945) tested 1,280 members of the Armed Forces in a forward area; 15 female nurses, 50 British officers, 100 British Other Ranks and 1,090 Indian Other Ranks formed the entire group. The percentages of persons with impaired dark adaptation were 13.3, 10.1 and 29.2 respectively. To show that this was probably due to vitamin A deficiency, vitaminized oil was fed to 43 selected persons; 25 of these showed improvement within a week and the night vision of 18 returned to normal.

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## CHAPTER XI

### VITAMINS B

A LARGE NUMBER OF Indian foodstuffs have been examined by various workers for vitamins of the B-complex. For this purpose the well known chemical methods have been used. In certain instances modifications and procedural refinements have been introduced. Thus Swaminathan (1938, 1940, 1941, 1942 and 1944) developed methods of estimation for thiamine, riboflavin, nicotinic acid and pyridoxine. Bhagvat (1943) has described the advantages of using a suspension of washed pig's intestinal mucosa for enzymic digestion of the test material, the product of this reaction, according to her, is also suitable for estimation of nicotinic acid and thiamine. Mukundan and Rama Sastri (1948) found that treatment of urine with basic lead acetate successfully removed substances interfering with thiochrome estimation. The following papers contain information about the methods used and the results obtained with different foodstuffs: Aykroyd and Swaminathan (1940), Passmore and Sundararajan (1941), Giri and Naganna (1941), Khorana, Sarma and Giri (1942), Rao, Ramachandran and Rau (1942), Daver and Ahmed (1944), Swaminathan (1946) and Bhagvat (1946).

Murthy (1937) compared the biological and fluorometric methods of riboflavin estimation in certain foodstuffs and obtained close agreement between the two. Sarma (1944) utilized the growth response of rice moth larvae (*Corcyra cephalonica* St) on pyridoxine deficient diets to determine the pyridoxine content of certain foodstuffs and reported good agreement with

the results obtained by Swaminathan's chemical method. Chitre and Desai (1949) report that not all the nicotinic acid found in foodstuffs is physiologically available. They found that only 26.1 to 54.2 per cent of nicotinic acid of certain foodstuffs was excreted in urine by niacin depleted rats. On the other hand, if pure nicotinic acid in equivalent quantity was fed the excretion was 41 to 77 per cent. They could recover after five hours from the intestinal contents of the rats an appreciable amount of nicotinic acid originating in foodstuffs like rice and pulses (Bengal gram, red gram,) whereas an equivalent quantity of pure nicotinic acid administered orally was completely absorbed within the same time. Chitre and Desai (1949 a) have described a method for the determination of total and available nicotinic acid. The observations reported by the above authors lend further support to those of Bhagvat and Misra (1946) reported three years earlier. On comparing the effects of acid and enzymic hydrolysis of foodstuffs on the liberation of nicotinic acid the latter had suggested that nicotinic acid set free by enzymic digestion represented the biologically available fraction.

Bhagvat and Devi (1944 a) have described the presence of an antithiamine factor in certain cereals, pulses and oilseeds. Its presence was suspected on account of the variable recovery of thiamine when added to the above foodstuffs. The factor can be extracted with chloroform water within a few minutes, is thermolabile and on dialysis separable into (a) thermolabile and nondialysable and (b) thermostable and dialysable fractions, both of which singly proved inactive. The action of the factor was probably nonenzymic since the activity was independent



of pH between 3 and 7. The product formed by the action of the factor on thiamine was biologically inactive in thiamine deficient rats and pigeons. On the other hand it was utilized by mosquito (*Aedes albopictus*) larvae for growth (Bhagvat and Devi, 1944 b). The authors believe (1944 c) that the factor present in cereals, pulses and oilseeds is different from the antithiamine factor of carp since the latter shows in action characteristics which indicate that it is an enzyme.

### Metabolic and other Studies

There are few observations on the metabolism of thiamine and riboflavin. Ahmad and Guha (1939) assayed the thiamine in human urine by rat growth method. They found in four healthy subjects a urinary excretion of 84 to 228  $\mu$ g. thiamine per 24 hours on an intake of 1200 to 1245  $\mu$ g. When the dietary intake was reduced to 600 to 800  $\mu$ g. a day, the excretion fell to 26 to 38  $\mu$ g./day within 5 to 6 days. It again increased equally rapidly when the higher level of intake was restored. Rama Sastri, Mukundan, and Patwardhan (1950) have observed the urinary thiamine excretion in fifteen normal men to vary between 66 to 1,200  $\mu$ g. on intakes varying from 0.8 to 1.39 mg./day. The level of excretion bore no relation to the level of intake. The fasting one hour excretion ranged from 3.4 to 90  $\mu$ g., but about 80 per cent of the observations fell in between 5 and 20  $\mu$ g. The authors found a correlation between the excretion in fasting one hour sample and that in the 24 hour specimen. The test dose returns were very variable ranging from 3 to 55 per cent and were independent of the level of intake and the pretest dose excretion. The authors could not find any correlation between

thiamine excretion and the non-fat calories in the diet.

The pyruvic acid (P) content of blood in these fifteen subjects ranged from 0.5 to 1.28 mg./100 c.c. (average 0.9 mg./100 c.c.) and lactic acid (L) in blood of 8 subjects was between 5.95 to 14.88 mg./100 c.c. The average P : L ratio was 1 : 9. All these values fall within the reported normal limits [Bueding and Wortis (1940), Golberg and Gillman (1943), Goldsmith (1948).]

All the above subjects were apparently healthy and showed values of urinary thiamine and blood pyruvic acid within normal limits. Hence it appeared that their thiamine intake was satisfactory for diets providing between 1,660 to 3,500 calories per day. In view of the above the authors suggest that the daily allowance of 1 to 2 mg. thiamine recommended by the Nutrition Advisory Committee in 1944 seems to be adequate.

Mukundan (1949) has also collected data for urinary thiamine and blood pyruvic and lactic acids of 25 girls of 4 to 14 years in age. In a nutrition survey, eighteen of these girls were graded very poor; their thiamine excretion gave an average value of 371  $\mu$ g, as compared with 386  $\mu$ g. found in children graded good. The pyruvic and lactic acid values and P : L ratios were 0.96 and 11.51 mg./100 c.c. and 1 : 11.9 in the "very poor" group and 1.16 and 14.01 mg./100 c.c. and 1 : 12 in the "good" group. There were no signs or symptoms of thiamine deficiency in any of the children belonging to the former group.

Mukundan, Rama Sastri and Patwardhan (*loc. cit*) observed in fifteen subjects referred to above a urinary riboflavin excretion ranging from 343 to

4.401  $\mu$ g. per 24 hours on a daily intake of 0.76 to 1.91 mg. The daily urinary excretion of riboflavin bore no relation to the intake. The test dose returned after the administration of 1 mg. riboflavin varied from 4 to 85 per cent and also had no relation to the pretest dose level of riboflavin excretion. Large amounts of riboflavin appeared to be synthesized in the gastrointestinal tract. It was observed that when diets contained large amounts of carbohydrates and relatively small amounts of fat and proteins, more riboflavin appeared in urine. When quantities of either fat, protein or both were increased, riboflavin in urine decreased, a fact which has also been observed by Rama Sastri (1949) in his riboflavin excretion studies on children 7 to 15 years of age. This result can be explained on the assumption that a better utilization of available riboflavin or a suppression of the microbial synthetic activity takes place under certain conditions. Whether one or both of these contribute to the observed alterations in riboflavin excretion is not yet clear. It is likely that both these factors may operate. Elvehjem (1946) has shown that in rats the severity of riboflavin deficiency could be increased by isocaloric substitution of fat for a large proportion of dextrin in a dextrin rich diet. The relationship between protein intake and riboflavin excretion has been observed by other authors [Sarett, Klein and Perlzweig (1942); Oldham, Lounds and Porter (1947); Czaczkes and Guggenheim (1946)]. It should be possible in future studies separately to assess the effects of dietary composition on (a) biosynthesis and (b) utilization of riboflavin.

Swaminathan (1939) found that healthy Indian adults (ten subjects) excreted 2.1 to 8.9 mg. nicotinic acid per 24 hours. Kochar (1941) observed in 6

subjects a urinary excretion ranging between 3.0 to 10.8 mg. per 24 hours and found that after oral administration of nicotinic acid ten to twenty per cent of the dose was excreted within the first three hours. On a dose of 100 mg. Swaminathan (*loc. cit*) reported an excretion of about 20-28 per cent during 24 hours. De and Banerjee (1948) found five subjects to excrete 3.7 to 7.9 mg. total nicotinic acid on an intake of 3.2 mg. per day. According to these authors a dose of 500 mg. nicotinamide resulted in 21 per cent of the dose being excreted in 24 hours. The authors suggest that the intake was adequate.

Kochar has reported nicotinic acid values in the blood of 41 young adults, the average being  $367.7 \pm 129 \mu\text{g}/100 \text{ c.c.}$

Not much work has been done in India on the metabolism or requirement of other members of the B-complex. Their importance in human nutrition as indicated by certain recent clinical observations discussed elsewhere should, however, attract the attention of workers in India.

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## CHAPTER XII

### VITAMIN C

MOST OF THE common Indian foodstuffs have been investigated for their vitamin C content. Before Tillmans' method of ascorbic acid estimation became well known a few attempts had been made to evaluate the antiscorbutic potency of Indian foodstuffs (Wats and Eyles, 1931 and 1932) by biological procedures. The easier chemical method has been used in all subsequent investigations although on a few occasions the comparison of chemical and biological methods has been attempted. Ahmad (1935) gives values for 65 foodstuffs and Ranganathan (1935) for over 100 foodstuffs. Biswas and Das (1939) reported that the ascorbic acid content of Indian chillies (*Capsicum annum*) increases on ripening. A significant finding is that even when the chillies are dried they retain a goodly proportion of ascorbic acid. Giri (1939) found that Indian gooseberry (*Phyllanthus embelica* Rom.) was an unusually rich source of ascorbic acid, containing about 7 mg. per gram of fresh pulp. On drying, there is a 50 per cent loss in vitamin content (Ranganathan 1942). The dry powder, when stored in bottles at room temperature, loses 50 per cent of its activity within six months and 80 per cent after 12 months. On the other hand, Mitra and Ghose (1942) reported a loss of only 66 per cent in one year. They, however, found that when stored in a refrigerator the loss in a year was approximately 20 per cent. The researches on Indian gooseberry had a topical interest, for in the early years of World War II, tablets and lozenges made from dried and powdered

fruit were extensively used for the Indian Armed Forces as a source of vitamin C. With the advent and easy availability of synthetic ascorbic acid the natural source was abandoned.

Another common foodstuff rich in ascorbic acid is drumstick (*Moringa oleifera*) leaf; values as high as 1.14 per cent on fresh weight have been reported (Panse and Sreenivasan, 1945). There are other edible leaves like amaranth, coriander, ipomea, etc., which are grown in plenty and are cheap in price. Certain popular fruits like guava, mango, papayà, etc., when they are in season would supplement vitamin C in Indian diets. It must be said, however, that the bulk of the Indian population relies on cheap green leafy vegetables for dietary supplies of ascorbic acid. Certain legumes and pulses on germination give rise to appreciable quantities of ascorbic acid [Bhagvat and Rao (1942 a and b), De and Barai (1949)]. Mere soaking in water does not cause the appearance of vitamin C; for it to do so the grain has to germinate. The maximum increase in ascorbic acid content can be demonstrated within 30–48 hours after the start of germination, the value then remains stationary for three to four days. As germination proceeds the ascorbic acid increases in the sprout and decreases in the cotyledons. The ungerminated dry gram (pulse in husk) contains 1 to 9 mg. ascorbic acid per 100 gm. Bhagvat and Rao (1942 b) later confirmed the presence of vitamin C in dry ungerminated Bengal gram by its antiscorbutic activity in guinea pigs.

Chakraborty and Roy (1936) reported an average urinary excretion of 9.65 mg. ascorbic acid per 24 hours in two subjects. They made an interesting observation that when rice in the diet was replaced

by equal quantities of fat or protein the ascorbic acid excretion rose by 50 per cent only to return to the original level after the subjects were returned to their usual carbohydrate rich diet. Ahmad (1935) had also observed that the indophenol reducing property of urine was increased on a high protein diet. Chopra and Roy (1936) suggested that all of the indophenol reducing capacity of urine was not necessarily due to ascorbic acid, particularly the increase on high protein diets might be due to the presence of reducing substances other than ascorbic acid. In their hands biological tests proved inconclusive.

Ranganathan and Sankaran (1937) determined the urinary ascorbic acid in 24 residents of Coonoor and Wellington belonging to different age groups. They observed values ranging from 1.2 mg. to 9.38 mg. per 24 hours with an average of 4.75 mg. Four young adults excreted between 7.6 and 9.4 mg. per day, 4 out of 5 children (5 to 13 years) between 1.2 to 2.5 mg. and 4 persons over 60 years between 1.7 to 5.3 mg. The excretion in 11 pregnant and lactating women was 1.35 to 7.72 mg. per day. All the above figures are low compared to those given by 5 Sikh and 5 British soldiers in their series in whom the urinary ascorbic acid varied from 10.8 to 27 mg. with an average of 18 mg. In these latter, a test dose of 300 mg. ascorbic acid resulted in a return of 3.7 to 8 per cent of the amount administered, only in one subject the test dose return was 44 per cent. In the other subjects test dose returns were extremely poor. In subjects of both the above groups load tests gave indications of unsaturation. Basu and Ray (1940 a and b) found in 17 middle class Bengalee boys a urinary excretion of ascorbic acid from 1.5 to 55 mg./day. Most of their subjects required 1,000 to 2,000

mg. for saturation. These authors reported that the excretion level was not necessarily indicative of unsaturation, for in two subjects, both excreting 11.8 mg. per day, they found that whereas one reacted to test dose within 24 hours like a saturated subject, the other required seven days' continuous dosage before there was an increase in the urinary excretion of ascorbic acid. These same authors (1940c) have reported excretion of 2.4 to 19.0 mg. per day in six adult subjects. Although the above observations are comparatively few to permit generalisations about the state of vitamin C nutrition of Indians being made, it will not be surprising if similar low figures are reported from other parts of India. The common Indian diets are deficient in vitamin C and a low urinary excretion of ascorbic acid should be expected.

Baksh, Kochar and Malik (1940) attempted to determine the vitamin C nutrition by the intradermal dye test. They raised a wheal on the forearm by injection of 40 mg. per cent (w/v) solution of the dye and determined the decolorisation time. The average value in 140 college students was found to be  $8.2 \pm 4.2$  min. The plasma vitamin C was  $1.19 \pm 0.3$  mg. per 100 c.c. The authors observed some correlation between the ascorbic acid content of plasma and the decolorisation time. They were, however, not satisfied about the reliability of the test. Banerjee and Guha (1939) observed in 41 normal Bengalee adults an average decolorisation time of 4 min. 53 sec.  $\pm$  1 min. 48 sec. In 9 other subjects decolorisation time was found to decrease after administration of 700 mg. ascorbic acid.

In a subsequent investigation Banerjee and Guha (1940) report an average decolorisation time of 85-95 seconds after saturation in six human subjects. Rotter (1939) had suggested that a decolorisation



time of five minutes indicated saturation, the above authors believe that the normal decolorisation time in saturated individuals must be as short as reported by them. A further detailed study is also reported by Banerjee (1944). He found that between the tests performed on the same individual on different days, on different forearms or two experiments simultaneously on one forearm there were no significant differences. In a survey of hostel diets, when diets were poor in ascorbic acid the decolorisation time was found significantly longer than in another season when vegetables were plenty and hence vitamin C in the diet adequate. It must be admitted, however, that in spite of the above satisfactory reports, the intra-dermal test has not been much used in India for the assessment of vitamin C nutrition. In fact not much work on the subject itself has been reported.

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## CHAPTER XIII

### BASAL METABOLISM

IN INDIA, the subject of basal metabolism first attracted attention in the middle twenties. This was followed by a spurt of activity which lasted nearly twenty years. Although it cannot be said that sufficient information has accumulated, the subject appears to have somehow failed to interest the Indian workers any more. It is a pity for so much work remains to be done.

Mukherjee (1926) reported the observations made on 15 subjects in Calcutta and in the following year Sokhey (1927) published the results of his preliminary work from Bombay. Observations on 100 male prisoners at Lucknow were reported by Banerji (1931) and up till 1940 papers continued to appear from various laboratories in India giving a general picture of the level of B.M.R. of Indian adults and in a few cases of children as well. 432 men from 15 to 75 years in age and 191 women from 15 to 55 years have been subjected to B.M.R. tests. 87 boys and 16 girls, both between 6 to 16 years, have also been studied. The results of all the above observations have been summarised in Table XIII. (*see pages 122, 123*)

The various averages reported by all the workers range from 34.3 to 36.7 Cals/m<sup>2</sup>/hr. for adult males and 30.9 to 35.1 for adult females. The average ages being 22 to 31 for males and 21 to 22 for females. The first thing that becomes clear, therefore, is that the age group for which sufficient data are available is extremely restricted. Some of the workers have omitted the details concerning age, height and weight.

etc., making it practically impossible to give a complete picture of all the studies on B.M.R. carried out in India.

The values for B.M.R. given above are lower than the current accepted standards in the western countries only two of which, viz., Harris and Benedict and Aub and DuBois have invariably been quoted in the Indian literature. Bose and De (1930) were the only workers who found that the B.M.R. of Indians came up to these two standards, their values ranging within +5 and -5 per cent of these standards. All the rest have found distinctly lower values both for men and women. It is difficult to offer any explanation for these unconfirmed observations of Bose and De and hence in the following discussion no further reference will be made to them. Another useful contribution which will have to be unfortunately omitted from discussion is that by Banerji (1931) already referred to. This author has given no figures for heat production, but only the deviation from certain standards which also he has left unnamed.

**B. M. R. of Adult Males:** The difference in the extreme average values reported by any two authors is 2.4 Cals/m<sup>2</sup>/hr. thus giving an error of 6.54 per cent on the highest average of 36.7 reported by Rahman (1939). Patwardhan (1944) has reviewed the work done in India on the subject of B. M. R. and has examined the reasons for these differences. He observes that they were due to (a) different techniques adopted to measure the B.M.R. and (b) difference in procedure followed to reach the average values. Most workers have used the Benedict-Roth type of apparatus of different manufacture and hence the minor variations in technique. A few have used the Douglas bag technique. Whereas

TABLE XIII—THE RESULTS OF INVESTIGATIONS

Authors and year	Place	Number of Subjects	Average Age	Nude Wt. Kg.	Standing height	Sitting height cms.
MEN						
1 Mukherjee (1926)	Calcutta	15	...	...	...	...
2 Mukherjee and Gupta (1931)	„	18	25	52.4	168	...
3 Banerji (1931)	Lucknow	100	...	...	...	...
4 Krishnan and Vareed (1932)	Madras	58	...	...	...	...
5 Bose & De (1934)	Calcutta	30	...	...	...	...
6 Rahman (1936)	Hyderabad (Dn.)	32	22	54.2	169.3	88.3
7 Rajagopal (1938)	Coonoor	26	31	56.0	168.0	84.0
8 Niyogi <i>et al</i> (1939)	Bombay	24	22.5	52.5	166.0	86.2
9 Sokhey & Malankar (1939)	„	60	26.2	55.5	167.9	...
10 Ahmed <i>et al</i> (1938)	Calcutta	9	27	60.3	166.7	...
11 Khanna & Manchanda (1946)	Lahore	60	...	...	...	...

## WOMEN

1	Mason and Benedict (1931)	Madras	27	Ta 21	44.9	154	78.2
			17	M 21	45.7	156	79.3
			6	Te 22	43.5	153	77.6
2	Krishnan and Vareed (1932)	„	15	...	...	...	...
3	Bose and De (1934)	Calcutta	30	...	...	...	...
4	Niyogi <i>et al</i> (1939)	Bombay	52	22	44.7	152.7	79.6
5	Khanna & Manchanda (1946)	Lahore	...	...	...	...	...

Ta = Tamil,

$$M = M$$



## BASAL METABOLISM

## METABOLISM BY ALL WORKERS IN INDIA

Oxygen Consumption c. c. per minute	Heat Production			Deviation from		Type of Apparatus used
	Cals in 24 hours	Cals/Kg.	Cal/m <sup>2</sup> /hour	Harris and Benedict	Aub and DuBois	
MEN						
...	...	...	...	...	—14	...
186	1299	24.79	34.26	...	—13.3	Douglas Bag & gas analysis
...	...	...	...	...	...	"British Benedict"
...	...	...	34.8	—10.8	—12.0	Benedict-Roth
209	...	...	...	...	± 5 standard	Sanborn
206	1431	26.40	36.7	—6.8	—8.7	Sanborn
192	1329	23.57	34.4	—8.9	—12.5	Benedict-Roth type
187	1297	24.75	34.5	—11.7	—12.9	Sanborn
202.5	...	...	36.3	—5.3	—8.0	Tissot Gasometer and gas analysis
207.8	...	...	36.49	—5.79	—8.99	Douglas Bag & gas analysis
...	...	...	35.66	...	...	"

## WOMEN

150	1048	23.3	31.3	—17.4	—16.8	Benedict
151	1052	23.0	30.9	—16.1	—18.2	
148	1033	23.7	31.3	—17.8	—15.8	
...	...	...	31.0	—18.2	—16.2	Portable Benedict-Roth
...	...	...	...	...	± 5 standard	Sanborn
152	1055	24.0	32.05	—15.9	—13.8	Sanborn
...	...	...	35.11	...	...	Douglas Bag & gas analysis

a Telugu Women.

those who have used the former type have assumed the respiratory quotient to be 0.82, the latter have calculated B.M.R. from the actual R.Q. observed by them. This has varied from 0.83 to 0.84 and calculations based on it will give slightly higher figures by about 1 per cent. The difference in technique, therefore, is responsible for a very small fraction of the difference. On the other hand the major difference has resulted owing to the second reason mentioned above. Rajagopal (1938) has used the lowest values for calculating averages; Niyogi, Patwardhan and Mordecai (1939) have used the mean of the two lowest values (not differing by more than 5 per cent) for the averages. Sokhey and Malandkar (1939) have taken the first reading only into consideration. It is, therefore, natural to expect the variations observed in the different experiments. With all this, however, the difference of  $2.4 \text{ Cal/m}^2/\text{hr.}$  does not appear to be large enough to invalidate the result of any of the workers.

**B. M. R. of Women:** In the series on women, only one set of observations by Khanna and Manchanda (1946) stands out different from all the others. These authors find an average of  $35.1 \text{ Cal/m}^2/\text{hr.}$  whereas reports of other workers give values between  $30.9$  and  $32.1 \text{ Cals/m}^2/\text{hr.}$  Unless confirmation is forthcoming for the observations of Khanna and Manchanda it will be right to assume that the B.M.R. of women in India lies within the range of  $30.9 - 32.1 \text{ Cals/m}^2/\text{hr.}$  Further it must be mentioned that Khanna and Manchanda report average value of  $35.66 \text{ Cals/m}^2/\text{hr.}$  for men in the same series. The observation that there is little difference between the B.M.R. values for men ( $35.66 \text{ Cals.}$ ) and for women ( $35.11 \text{ Cals.}$ ) is at variance with the results obtained by all the workers in India and abroad.

There is little doubt, therefore, that the reported B.M.R. of Indian men is lower than the Harris and Benedict and Aub and DuBois standards for Americans. The B.M.R. of Indian men is lower by 5.3 to 11.7 per cent as compared with the above standards and the B.M.R. of women deviates by—15.9 to—18.2 per cent with respect to Harris and Benedict standards and —13.8 to —18.2 per cent with respect to the Aub and DuBois standards. Attempts have been made by almost all the Indian authors to find an explanation for the low B.M.R. of their subjects. Among the possible or probable causes were mentioned (*a*) state of nourishment (*b*) low level of protein metabolism, (*c*) tropical climate, (*d*) state of muscular relaxation, (*e*) surface area and lastly (*f*) race. All these explanations have been critically examined by the author in his review entitled “Studies on Basal Metabolism” published in 1944. A short summary of the discussion examining the validity of arguments advanced is given below, for it is felt that the criticism then made is still valid.

(a) **State of Nourishment:** The men and women selected for most of the Indian studies on B.M.R. were drawn from (i) medical students (ii) staff of medical colleges and (iii) nursing staff of the hospitals. The subjects must, therefore, have belonged to middle and higher middle classes, for the class of population from which such personnel is drawn could hardly be otherwise. It is, therefore, most unlikely to find undernourishment in this class of persons. This was substantiated at least in the case of subjects from Bombay where a diet survey showed that their diets bordered on the luxurious. Further, the physical examination of the subjects prior to selection for the B.M.R. tests did not indicate a state of

undernourishment. If Pelidisi or Pirquet Index is any indication of the state of nutrition (which again is a doubtful assumption) the reported figures do not show any relation between it and the B.M.R. [Niyogi *et al* (1940)]. It is, therefore, a moot point whether the state of nutrition has been at all responsible for the low B.M.R. of Indian subjects.

(b) **Protein Metabolism:** Krishnan and Vareed (1932) observe that the subjects who were "definitely known to be" on a high protein diet gave higher B.M.R. than those "known to be" on low protein diet. There is no quantitative information in the paper to enable one to check the validity of the above observation. Sokhey and Malandkar (1939) were of the opinion that the low level of dietary protein in Indians was responsible for their low B.M.R. Here again the statement was unsupported by factual evidence. Niyogi, Patwardhan and Sirsat (1940) found by diet survey methods the protein intake of 10 of their subjects to be on an average 72 gm., although the average urinary nitrogen was only 6.25 gm. per 24 hours. The protein consumption levels reported for American medical students by Denis and Borgstrom (1924), Beard (1927) and Brooks (1929) were also of the same order. Further, controlled feeding experiments by Patwardhan, Rama Sastri, Mukundan and Tulpule (1950) and Karambelkar, Patwardhan and Sreenivasan (1950) have shown that low urinary nitrogen values of 5 to 6 gm. per 24 hours are obtained in Indians on an intake of 10 to 12 gm. nitrogen daily. The significance of this fact has been discussed elsewhere. It is, however, true that on the dietary pattern obtainable in India the level of protein metabolism as determined by the urinary nitrogen excretion appears to be low. Patwardhan

and his associates have further shown that by merely changing the pattern of protein mixture in the diet without altering the total protein intake one can bring about an increase in urinary nitrogen. This effect is marked when vegetable proteins are substituted to over 50 per cent by proteins of animal origin. The level of protein metabolism then appears to be higher, but the question whether such high level of protein metabolism would determine the level of B.M.R. has yet to be settled. Niyogi, Patwardhan and Sirsat (1940) have demonstrated that raising the level of protein metabolism for short periods by feeding extra protein did not raise the level of B.M.R. of ten subjects studied by them. On the other hand, there is an observation by Benedict, Kung and Wilson (1937) on 88 Chinese, 28 Manchus and others of the Mongolian race which indicates that the level of protein metabolism may not bear a direct relation to B.M.R. (as has been suggested by some Indian workers). These authors found that although the daily urine nitrogen in the Chinese and Manchu subjects was the same as or even higher than in Americans, the B.M.R. was distinctly lower. This evidence will have to be borne in mind in assessing the influence of the level of protein metabolism on B.M.R.

(c) **Muscular Relaxation:** Necheles (1930, 1932) found that in Chinese there was very little difference in the B.M.R. when awake or asleep. It had been demonstrated before (Talbot, 1925) that there is a fall of 10 per cent in B.M.R. when the subject is asleep. This difference is due to muscular relaxation during sleep, so that heat production is lower than when muscles are in tone during waking hours. Mason and Benedict (1931) made the suggestion that



one of the causes for the low B.M.R. of Indians was possibly the lower tone of muscles supposed to be due to the high environmental temperature of the tropics. Three years later these authors (1934) re-examined the question and found that under controlled conditions South Indian women during sleep showed a fall of 10 per cent in the B.M.R. They were also of the opinion that Necheles did not carry out his observations under ideal conditions essential for the study of B.M.R. during sleep and hence his interpretation of his own results was not valid.

(d) **Tropical Climate:** The role of climate in determining the level of B.M.R. has excited a great deal of controversy. De Almeida (1920) reported that B.M.R. of white men in Brazil was 16 per cent below the American standards. Haffekesbring and Borgstrom (1926) and Tilt and Waters (1935) investigating the B.M.R. of southern whites in the U.S.A. found values distinctly lower than Harris and Benedict and Aub DuBois averages. On the other hand, Williams and Benedict (1928) who followed the B.M.R. of whites going to the subtropical and humid climate of Chichen Itza, Yucatan, found it unaffected by climatic changes. Eight white male residents of Yucatan gave a value lower by—6.3 per cent (H & B) whereas females gave normal results.

Some light is thrown on this subject by the studies of Mason (1934, 1940). This author found the B.M.R. of 34 American women residents of Madras to be —7.9 per cent (H & B) and—12.5 per cent (A and DuB). Nine of these women were studied when they returned to the U.S.A., four showed an increase of 10 per cent over their Madras values whereas in five others there was no change. Three South Indian women were observed in India, the U.S.A. and

England. They showed a difference of 4.8 per cent only, the higher values being recorded in the temperate climate. Mason, therefore, is of the opinion that the tropical climate may be responsible for lowering B.M.R. by about 5 per cent. It is worthwhile to remember that the average B.M.R. in 64 of her subjects was lower than H & B standards by 17.1 per cent. Thus even granting that Mason's evaluation of the effect of tropical climate is approximately correct the above low values still require a satisfactory explanation. Mason made a very interesting observation on the American subjects whose B.M.R. were refractory to climatic change. She found that when these subjects moved from the U.S.A. to India, they showed an increase of 0.2 to 0.7°F. in mouth temperatures whereas those women who reacted by a fall in B.M.R. on migration to tropics did not show any alteration in their mouth temperature. Mason has recorded a high degree of correlation between B.M.R. and mouth temperature changes. In 1940 she was able to confirm these findings by following the B.M.R. and mouth temperature of 9 English and 3 American women in tropics and temperate regions. It, therefore, appears probable that two types exist, one reacting to tropical climate by a fall in B.M.R. and the other refractory. Martin (1930) did hint at the possibility of existence of such refractory types. Scott MacGregor and Loh (1941) later confirmed the existence of two such types by their observations on Europeans in Singapore.

(e) **Surface Area :** All the workers in India have used the formula of DuBois and DuBois (1916) or rather the nomogram prepared from it. DuBois (1936) had compared his height-weight formula with the linear formula and had reported good agreement.

The application of these formulae to other races was attempted by several workers. Takahira (1925) who compared the results obtained by paper mould method with the predictions of several different formulae (e.g. Meeh, Lissauer, Stoltzner and Miwa, Bourchard and DuBois and DuBois) found the best agreement with the height-weight formula of DuBois and DuBois, provided the constant C was changed to 72.46, for according to him C becomes larger when height decreases. Stevenson (1930) and Necheles and Loo (1932) found that in Chinese the height-weight formula gave higher results than the linear formula, whereas McLeod, Crofts and Benedict (1925) obtained lower results with the use of the height-weight formula. The above observations point to a maximum difference of 3 per cent which cannot account for the low B.M.R. observed in Indians.

(f) **Race:** It is true that significant differences on both sides of the Aub DuBois and Harris-Benedict standards have been recorded in different races in several parts of the world. The author hazards a guess that the reasons underlying these differences can only be explained adequately when we understand much more about the response of the organism to external environment than we do at the moment. There is no evidence to show that race modifies any other physiological function of the body or even the composition of body fluids which are primarily concerned in maintaining the internal environment constant. Hence it is difficult to conclude on insufficient evidence that racial origin would determine by itself the rate of energy production.

**Other Considerations:** It seems necessary, however, to point out one fact to which not sufficient attention has been paid. In accepting the Atwater

standards for the calorific value of oxygen we are assuming that the former would be applicable to the peculiar dietary pattern on which the population of the tropics subsists. It has been mentioned by Atwater himself that the values obtained by him were for the particular dietary patterns as they prevailed in Europe and America. Any gross alteration in the former would require re-investigation of the whole problem. It is not implied here that a recalculation of Atwater calorie co-efficients for oxygen would account for either the whole or a large part of the differences between the American and Indian B.M.R. values. It is suggested, however, that the problem needs careful consideration.

There are great many gaps in our knowledge about the B. M. R. of Indians. It is true that the investigations in different parts of India have revealed uniformity in the rate of energy expenditure in certain age groups. But our knowledge about B.M.R. of children and of older age groups of both sexes in India is fragmentary. It is essential that standards of B.M.R. covering a wide range of age groups are formulated, for it is clear that we cannot use the Harris and Benedict or Aub-DuBois or any other standards in the West for purposes of comparison. In the absence of relevant information, clinics and hospitals still continue to apply an arbitrary figure for deviation from the two best known standards. It cannot be denied that this state of affairs is highly unsatisfactory and ought to be soon remedied.

Apart from the fundamental considerations which determine the low basal energy expenditure of Indians, the problem of energy requirements is of utmost practical importance to this country where



an acute overall food shortage is now being felt. In the first instance it is necessary to find out the deficit in our total calorie supply. This itself is a formidable task for want of accurate information. Although we have an approximate idea of age and sex distribution of the population we have very little information about the proportion of population engaged in different levels of activity. The Indian estimates for total energy requirements of an average adult male engaged in eight hours of moderate physical activity are in the neighbourhood of 2,600 - 2,700 calories per day. Considering the average body weight of an Indian as 55 kg. the figure quoted above does not materially differ from an estimate recently made by the F. A. O. Committee on Calorie Requirements (1950). Even the latter has been arrived at on the basis of certain considerations which will have to be experimentally tested. It is, therefore, imperative not only to collect further data on B.M.R. of Indians of different age groups, but also to ascertain experimentally the cost of different types of work in terms of extra energy expended. Any estimate regarding the total energy requirements of Indians made in the present state of our knowledge will be more in the nature of guesswork than based on experimental observations. It is undoubted that energy metabolism presents very wide scope for investigation in India.

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## CHAPTER XIV

### DIETS

TO A CASUAL observer the dietaries of the people of the various regions in India appear to be materially different in composition and nutritive value. The differences which exist are, however, superficial and originate in the different methods of preparation of various foods and not so much in the average composition of the diet. It cannot be denied, however, that the former can materially alter the nutritive value of foods and thus bring about differences in diet which should be more deep seated than a mere enumeration of foodstuffs would indicate. But as the discussion at a later stage will show, cooking practices in India are generally not so drastic as to make significant variations in the nutritive value of foods consumed. Basically the dietary habits conform to a certain well known pattern.

The information on the Indian dietaries has been extensively collected only recently. The major constituents of the diets and their relative proportions were known for a fairly long time. These were taken into consideration when formulating the dietaries of armed forces or jail population by the authorities under the British regime. McCay (1910) refers to certain attempts made in the latter half of the nineteenth century to formulate dietaries for Indian prison population based on Indian foodstuffs and in accordance with the dietaries for similar groups in England. The basis for these attempts was the known chemical composition of foodstuffs with respect to proteins, fats, carbohydrates and minerals only. The

fact that in the formulation of diets these were not the only factors to be taken into account was even then realised as the following quotation will show. "A mere tabular statement of the several ingredients constituting the several diets would be of comparatively little value unless accompanied by a statement of their chemical composition; and, in order to judge of the comparative merits of dietaries so analysed, it is essential that a clear conception should be formed as to what particular portions are specially adapted to the nutrition of the body."

In 1881, Lewis again drew attention to the fact that no attempts had till then been made to study the food requirements of the inhabitants of India "undergoing laborious exertion". Prison and army diets being the particular responsibility of the government, the latter did make attempts to bring the dietary schedule in conformity with the prevailing practices in the country. Detailed information on this, however, was still wanting and in spite of McCay's intensive investigations on jail dietaries of Bengal and the United Provinces and those of Macnamara on Punjab jail dietaries, information on the dietaries of the general population remained only sketchy.

In 1923, Das studied the diets of students in a few boarding houses and in 1929 Banerji described in general diets in the United Provinces. The real activity in the field of dietary studies commenced after 1930. The fourth decade of the nineteenth century was particularly suitable for such activities. By then sufficient information on the role of vitamins and some other essential nutrients in nutrition had accumulated and it had become possible to assess scientifically the influence of diets on the occurrence of certain diseases in the general population. The

Health Organisation of the League of Nations (1936) had taken great pains to collect information on practical aspects of nutrition and in doing so had recommended certain standards of physiological requirements. These served useful purpose as international yardsticks for comparison of the nutrition of the people in relation to their dietaries. It was soon realised that these standards were not necessarily the last word on the subject; that they were capable of being revised in the light of further knowledge was also recognised. On the other hand, this fact itself prompted further studies on dietaries in regions where some work had already been done and initiated such work in regions where no information on diets existed. India came under the latter category. The interest of the countries in the South-East Asian Region was further aroused by the recommendations of the Intergovernmental Conference of Far Eastern Countries on Rural Hygiene which met at Bandoeng (Java) in 1937 under the auspices of the Health Organisation of the League of Nations.

In India, intense activity in the field of nutrition followed the above conference and one of the problems to which considerable attention was paid was the dietaries of the Indian people. The lack of trained personnel and facilities rendered intensive work difficult till much later. By 1938, however, 50 surveys in different parts of India had been carried out and the basis of information laid down. This was collected by Aykroyd and published in his 'Note on Results of Diet Surveys in India' in 1939. The work thus started was hampered by World War II, but by the end of 1945, 99 surveys were on record although several more have remained unpublished. The food shortage resulting from the war gave a fresh impetus



to this type of work and by 1949 nearly a thousand surveys had been made. It can now be said that a reasonable picture of Indian dietaries is available. It must be mentioned, however, that although the later surveys have provided more quantitative material they have not provided data which would require appreciable modification of the main conclusions drawn from the first 50 surveys conducted up to 1938.

The technique followed in these surveys has been more or less uniform. The information was collected by house to house visit by investigators who weighed every day the foodstuffs used for the preparation of meals. It has been the experience that in Indian houses, especially in those of the poor, plate wastage is negligible for the simple reason that the people, on account of their poverty, prefer to eat all that they prepare for meals. Hence there has been no need to allow for plate wastage. Then again, food left over from one meal is usually consumed at the next. If, therefore, the survey has lasted for a week, it includes all the food thus consumed. In practice, the surveys have covered periods of seven to twentyone days. The longer periods were preferred during the earlier period of surveys. During the last few years, however, most surveys have restricted themselves to seven to ten days.

A large number of surveys was devoted to the study of diets of the poor people from the rural areas, a few deal with low income groups in towns and still others with the diets of the middle class people. The groups of population dealt with cover agriculturists, agricultural labourers, Harijans and backward classes, aboriginal tribes, factory workers, clerks, petty tradesmen and middle class families of persons engaged in

numerous other walks of life. A great deal of work has also been done on institutional diets as well. Full details of all diet surveys will be found in Aykroyd's note referred to above and in a more recent publication on the subject by the Indian Council of Medical Research (1951). In Tables XIV and XV are given the results of a few sample surveys conducted on poor people in villages in different provinces and on middle class people in similar areas. The town population in the low income groups has strong roots in the country and on account of frequent migratory visits to villages it keeps up its habits materially unchanged particularly with regard to diets. This opinion has been confirmed by diet surveys carried out in towns and cities.

**Characteristics of Indian Diets :** The two peculiarities in the poor Indian diets which should strike even a casual observer are (a) the preponderance of cereals and (b) a marked deficiency in protective foodstuffs. Both of these features are common in the diets of poor people irrespective of which part of the country they live in or belong to. Small variations in the intake of individual foodstuffs may be found from province to province depending upon the price and relative abundance of any foodstuff, but the main pattern is undeniably similar in all the provinces. The dependence on cereals for energy purposes is exaggerated on account of the lack of other foodstuffs in the diet. Figure V shows the average (if calculating an average is permissible) poor Indian dietary obtaining in pre-war days.

The chart also contains information about the diets of middle class families from different parts of India. A glance at the combined chart shows that the middle class diet is relatively better in that it is

TABLE XIV—POOR INDIAN DIETARIES (1937—1942)

Foodstuff	Madras	Mysore	Baroda	Madhya Pradesh	Bengal	Orissa	Bihar	Uttar Pradesh	Punjab	Assam	Kashmir	Hyderabad
Rice	14	2.4	8.1	26.4	23.7	24.6	20.1	6.8	—	19	25.6	0.9
Wheat	—	—	—	0.2	—	—	—	17.0	22.4	—	1.3	2.0
Millets	6	24.9	11.3	—	—	—	—	—	—	—	—	22.1
Other Cereals	—	—	—	—	—	—	—	—	—	—	—	—
Pulses	1.4	2.1	4.0	1.1	1.4	0.9	0.6	2.4	2.7	1.0	0.6	1.7
Leafy Vegetables	0.3	1.3	—	1.5	0.8	0.3	2.0	1.6	3.3	0.2	5.2	0.2
Non-leafy Vegetables	3.4	0.9	2.6	3.1	9.2	7.7	0.5	2.3		3.4	1.5	0.8
Fruits	—	—	—	0.1	0.6	—	—	—	—	—	—	0.6
Oils and Ghee	0.5	0.1	0.7	0.2	0.6	0.1	0.1	0.5	0.4	0.3	0.9	0.6
Milk	—	1.9	2.3	0.2	3.1	—	0.1	6.4	2.4	0.5	2.2	2.1
Meat, Fish & Eggs	0.2	—	—	0.1	1.8	0.6	0.1	—	0.7	0.2	0.2	0.3
Sugar & Jaggery	—	—	—	—	—	—	—	0.2	—	—	0.5	0.6

IN OZ. PER CONSUMPTION UNITS

Foodstuff	Bengal	Bihar	Bombay	Gujarat	Madras	Punjab	
						Hindus	Muslims
Rice	10.2		8.2	3.6	13.3	1.5	4.1
Wheat	3.1	18.0	3.2	5.6	—	10.9	11.6
Millet	—		0.2	0.8	—	—	0.7
Other Cereals	—	—	—	—	0.8	4.5	—
Pulses	1.5	4.1	1.1	1.8	1.3	1.8	1.2
Leafy Vegetables	0.7	1.2	1.8	7.0	0.6	4.0	3.0
Non-leafy vegetables	11.6	5.9	3.2		3.9	4.5	3.0
Fruits	3.3	0.6	—	—	—	0.7	—
Oil and Ghee	2.2	1.8	2.9	2.6	1.3	1.3	1.4
Milk	11.0	4.1	7.6	10.6	10.5	10.7	7.0
Meat, Fish, Eggs	5.6	3.7	4.1	—	0.6	0.1	2.2
Sugar and Jaggery	—	0.6	1.9	1.8	1.8	2.2	1.1

IN OZ. PER CONSUMPTION UNITS

balanced to a certain extent whereas no such balance exists in the poor man's diet. In the middle class diets there is a larger proportion of fats, milk, flesh foods and sugar. All of them are costly items in the diet and this explains why they are not included in the poor man's dietary. It must be said that balancing of the diet on the part of medium or higher income groups is instinctive and not necessarily due to any studied attempts based on knowledge of nutritional science. Similar has been our experience in a study of dietaries of textile labour in Bombay; Mitra (1940) also finds that with the increase in income,

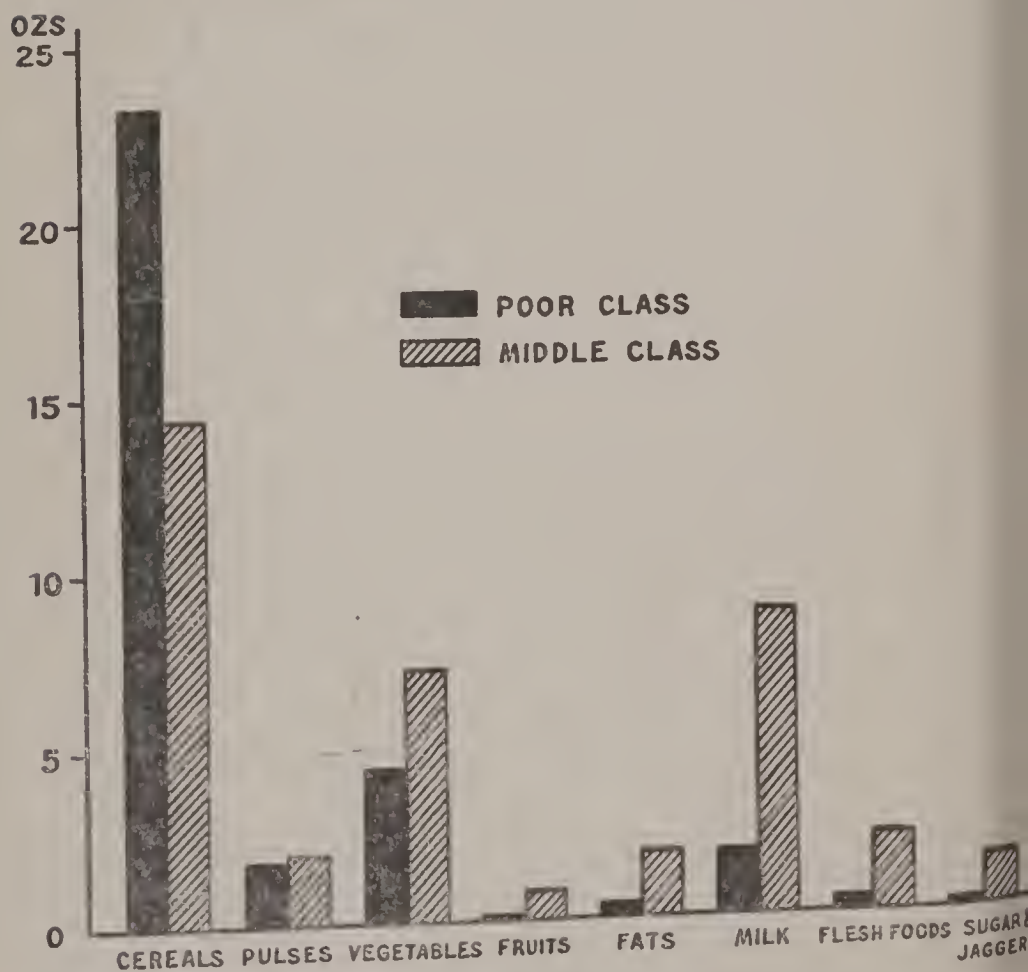


Figure V—Diets of average poor and middle class families.



the labourers in the iron and steel town of Jamshedpur adjust their dietaries in such a way as to ensure greater balance in the various foodstuffs consumed.

A description of the poor Indian dietaries cannot be complete without reference to the calorie intake and fat and protein content of their diets. The comments which are made below are based on a critical analysis of 139 diet surveys carried out in different parts of India during the years 1931 - 1942. Only two diet surveys were conducted in 1931, the remaining 137 were reported between 1937 - 1942. This period was characterized by the presence of a world depression in which the prices as also the incomes—particularly of agriculturists and wage-earners—were maintained at a low level. Although the second World War broke out in September 1939, the effect on prices and incomes was not felt in India, till the second half of 1942, i.e. when Japan had made a temporarily successful bid for power and was on the eastern frontier of India. It was only after this that the economic conditions in India started on a headlong career of inflation which as yet has shown little sign of being checked. The period of 1931-1942 is, therefore, one in which the dietary studies reveal the influence of poverty on the diets of the masses.

The material on 139 surveys has been collected partly from published papers and partly from some unpublished reports available to the author. In a few cases the information is not complete, e.g., some reports contain information on the number of families but not the number of individuals, others give the number of persons but make no mention of the number of families. There is, however, uniformity in the technique employed in almost all of these surveys, and the calculations of energy values and nutrients have

been based on tables contained in Health Bulletin No. 23. Thus the results are comparable (except for the two surveys conducted in 1931). The following table (Table XVI) gives the number of surveys according to the provinces and some of the larger princely states of the then undivided India.

TABLE XVI—DIET SURVEYS IN INDIA 1931-1942

PROVINCE OR STATE	Number of Surveys
Travancore	10
Madras	5
Mysore	2
Coorg	3
Bombay	6
Baroda	1
Hyderabad	28
Orissa	30
Central Provinces	6
Bengal	2
Assam	2
Bihar	23
United Provinces	2
Delhi Province	6
Punjab	13

These surveys provide material for over 3,250 families comprising over 14,000 persons. Since the area covered—albeit unevenly—by these surveys has been country wide, the interpretation of results has great value. One justification for using these data is that other writers have made comments on dietary habits on much less quantitative material available to them.

The above number of diet surveys includes only those that were carried out on low income groups. These latter consist mainly of cultivators, agricultural labourers, and agriculturists. In a few instances, however, the low income groups in urban areas, such as petty tradesmen, industrial labourers and others have been surveyed. It is felt that although the information obtained from the different parts of India is uneven in its incidence, it does represent a cross section of the dietary habits of the Indian population. In any case the surveys provide a factual basis for the conclusions drawn about the diets in India. All the results are expressed in terms of "man value" or "consumption units" calculated by the use of conversion factors as given in Health Bulletin No. 23. It is realized that this method of conversion is not free from fallacies. The requirements of nutrients differ according to age, sex and physiological stresses like pregnancy and lactation. No uniform method of conversion into "man value" is likely to take care of the above differences. However, it is felt that since the present method has been largely adopted in India, the results obtained are capable of interpretation, provided its limitations are borne in mind.

**Calories:** The average calorie intake calculated from the available data amounts to 2,560 calories per consumption unit per day. In 92 surveys the calorie intake was between 2,000 - 3,000 calories and in 28 surveys it was between 3,000 - 4,300 calories while 17 surveys have recorded intakes of 1,100 - 1,500. These last are from extremely poor people who were no doubt on starvation diets. The average value of 2,560 is low but not very low when one considers that it includes men and women of all ages engaged in

different states of physical activity and children of varying ages. Aykroyd's estimate of energy requirement for an average Indian is 2,500 - 2,600 calories.

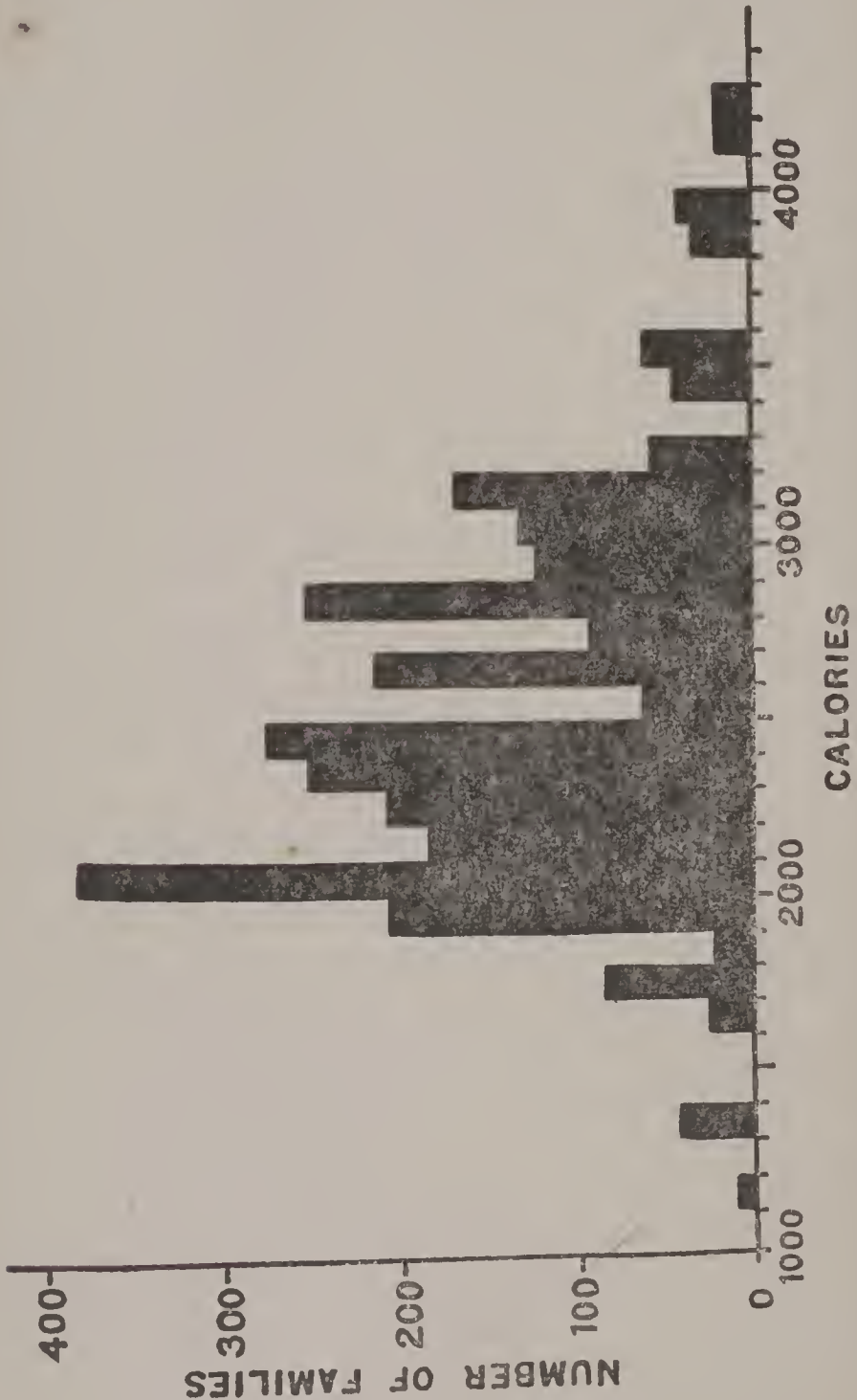


Figure VI—Frequency distribution of calorie intake observed in diet surveys on 3,209 families from different regions of India.

is noteworthy that the lowest intakes have been recorded in surveys in Travancore and the highest in the Punjab. The distribution of the intermediate values for calorie intake is more or less even throughout the country. It must also be mentioned that between 70-80 per cent calories, and in some instances more, are derived from cereals and pulses, the latter contributing only a small fraction of this portion. The distribution of calorie intake in these surveys is shown in Figure VI.

**Proteins:** The figures for proteins intake were available in 132 surveys. These yield an average value of 73 gm. per consumption unit per day. In 8 of these surveys the protein intake has been recorded between 51 and 100 gm. In 17 surveys, protein consumed per day has been shown to be between 20 and 50 gm. (Figure VII). Several of the latter values correspond to those surveys in which extremely low calorie intakes were recorded. The bulk of the protein in Indian diets is derived from cereals, pulses and other vegetable sources. The relative figures for animal protein consumption are not available in all the surveys. About 40 such surveys make a mention of animal protein intake, and from these it would appear that on an average about 9 per cent of the total protein in the diet could be of animal origin. This is probably an optimistic estimate; if the information was available from all the 132 surveys instead of only 38, it is likely that this figure for animal protein would have been very much lower. That this statement is substantially correct can be seen by the general pattern of the diet which contains extremely small quantities of milk and milk products and flesh foods. Thus although it cannot be said that the average poor man's diet in India is deficient or



markedly low in proteins, it cannot be denied that proteins of high biological value are possibly lacking.

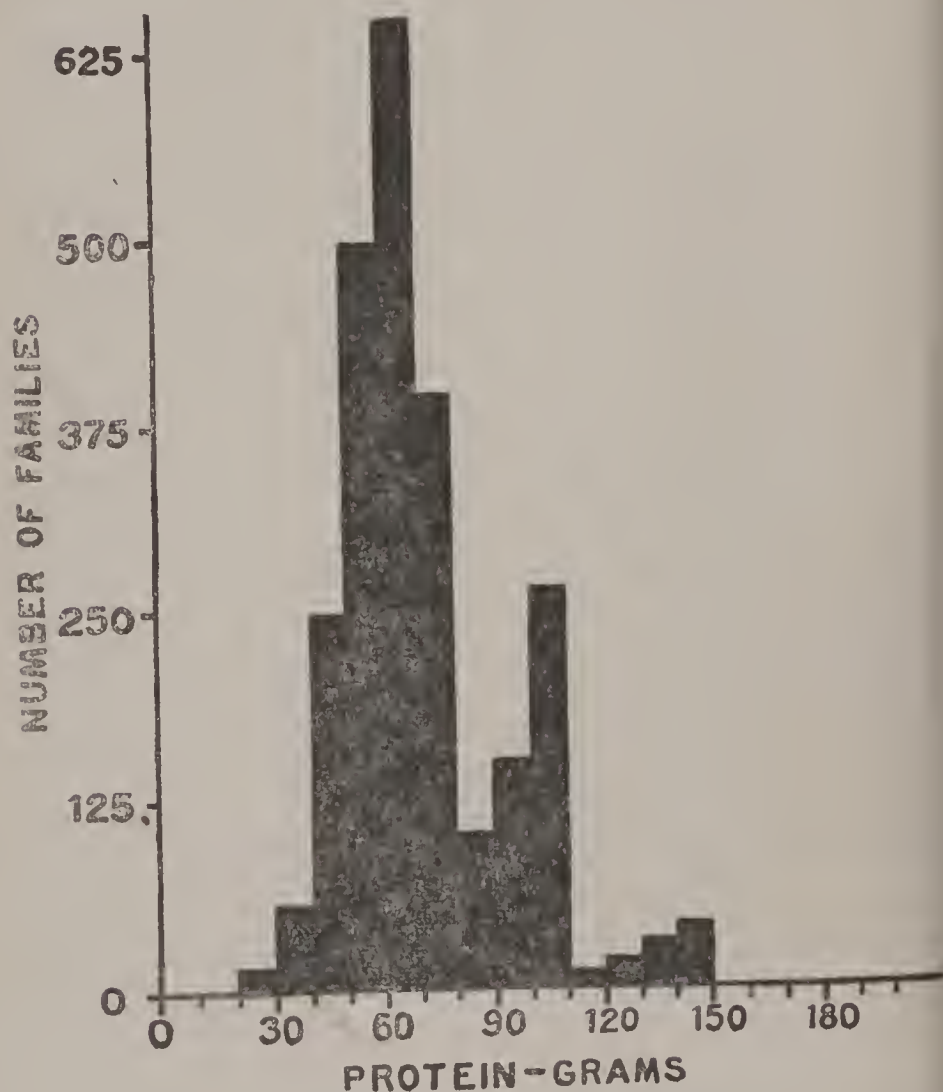


Figure VII—Frequency distribution of protein intake observed in diet surveys on 2,514 families from different regions of India.

**Fats:** The average intake of fats calculated from the figures available in 127 of the above surveys amounts to 23.5 gm. per consumption unit per day.

The frequency distribution of fat consumption as ascertained in these surveys is given in Figure VIII.

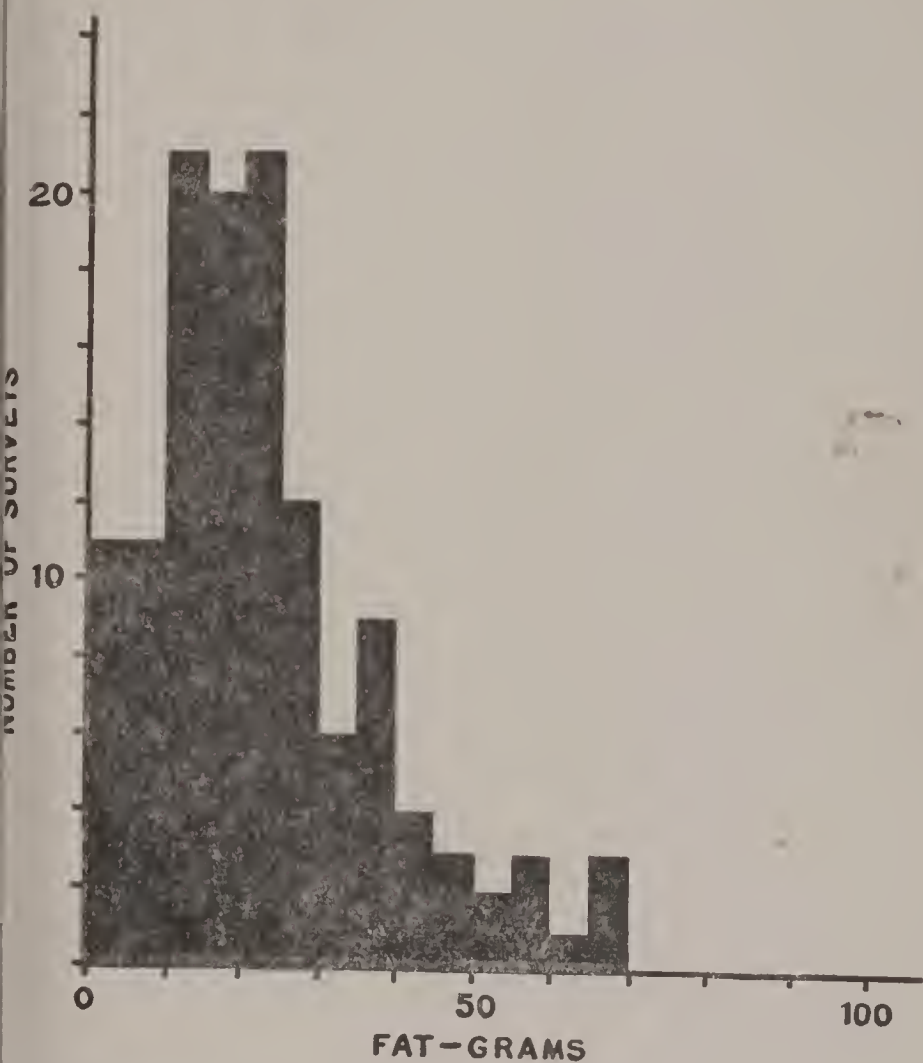


Figure VIII—Frequency distribution of fat consumption observed in 127 diet surveys.

Most of the fat is derived from the vegetable oils utilized for cooking purposes and a minor indeterminate fraction derived from milk and milk products. In absence of any reliable information regarding the requirements of fat, it would be hasty to pronounce

this figure as low. Considering, however, that an increased intake of nutritious foods such as milk and milk products or meat, fish and eggs improves the fat content of diets, it must be admitted that the observed average fat intake gives an additional proof, if any is needed, of the poor quality of the diet consumed by the Indian masses.

A similar analysis to obtain figures for mineral and vitamin intake has not been attempted. Firstly, not enough detailed information is available in most of the diet surveys and secondly, even if it were available, the enormous amount of calculation work involved would have merely confirmed the conclusions about the dietary defects, which are self-evident in the Indian diets. A few comments on both these classes of nutrients will not be out of place even though the statements made will not be supported by figures.

**Minerals:** The relation between minerals in diets and the health of the population will be described elsewhere in this book. In North India in the Sub-Himalayan region endemic goitre is present and its association with iodine deficiency has been demonstrated. In Kangra Valley in the Punjab, the diets are deficient in calcium and phosphorous together with other nutrients and their effect on the incidence of rickets, tetany and osteomalacia is referred to in another chapter. The occurrence of microcytic hypochromic anæmia responding to iron therapy demonstrated in labour recruited for Assam tea gardens and more recently in recruits drafted for the Indian Army during 1939-1945 gives an indication of iron deficiency in the diets. The considerably low consumption of vegetables and fruits or milk is responsible for deficiency in mineral components of the

diet. The large amounts of cereals provide phosphorous chiefly in the form of phytin, more than half of which is unavailable to the human body and which also interferes with the absorption of calcium and magnesium due to the insolubility of the salt. All these considerations lead to the conclusion that the poor Indian diets must have a low grade deficiency of more than one mineral nutrient.

**Vitamins:** For reasons mentioned in the last paragraph, the vitamin content of the poor Indian diets must also be considered to be inadequate. This may not be true of all diets. In rice diets a possibility of thiamine deficiency does exist but thanks to the practice of parboiling, the danger is considerably reduced. Beri beri has been recorded in those rice eating regions where people do not consume parboiled rice. In wheat or millet eating people of India, thiamine deficiency is not likely to occur and has been observed only rarely. The evidence regarding riboflavin and nicotinic acid deficiency is discussed elsewhere. One of the commonest deficiencies is expected to be that of vitamin A. The diets do not contain enough preformed vitamin A and in a majority of cases the carotenoid content is also low. Now and then, the specific deficiency symptoms are seen in the child population. On the whole, however, it will be safe to assume that in this respect also there is a mild chronic deficiency existing among the population.

**The effect of War and Post-War conditions on Indian Diets:** As mentioned earlier in this Chapter the effects of World War II began to be felt in the latter half of the year 1942. Since then the prices of foodstuffs have continuously risen together with those of other commodities and although the incomes have

also increased, the latter have not been commensurate with the general increase in prices. The price levels of different foodstuffs have increased from 400 to 500 per cent over those existing in 1939. This fact coupled with the general shortage of food supplies has had an adverse influence on the diets of the poor people. Recent diet surveys have shown, however, that in rural areas the same types of diets are being consumed as before the war, there being little evidence of cereal shortage. On the other hand in urban and semi-urban areas calorie intake has decreased owing to the fact that cereals are rationed and the amounts procured by different provincial governments plus those allotted by the Central Government are not enough to provide even 14 ounces per head as recommended by the Nutrition Advisory Committee. Substitute foodstuffs are also in short supply and are costly. There has been, therefore, an undoubted deterioration in the quality of diets. This deterioration has been more marked among the lower middle and middle classes of population, particularly the people with salaried incomes, for their incomes have not proportionately increased. The income of industrial labour has gone up by 100 to 200 per cent over the pre-war level, whereas the maximum increase in salaried workers of the middle class cannot be said to have exceeded 20 per cent.

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## CHAPTER XV

### NUTRITION SURVEYS

ALONG WITH the diet surveys reported in the last chapter, nutrition surveys were also carried out simultaneously. The criteria which were used in evaluating the nutritional status between the years 1937 and 1942 were rather crude. The emphasis was mainly on heights and weights, general appearance and presence of signs and symptoms of deficiency states. The newer biophysical and biochemical techniques were then not sufficiently developed and known to Indian workers. Even where the investigators were aware of them, their use was precluded on account of the lack of equipment and staff. In a few surveys the applicability of indices like Franzen and Palmer's A.C.H. index, Knudsen-Schiøtz index, etc. was tested. On the whole, however, these indices did not find much favour with the field investigation staff in India.

The records of most nutrition surveys cover more or less the bare essentials which are often insufficient for the object of proper assessment of nutritional status. In spite of these handicaps, however, the information which has been collected has proved of value in comparing certain physical characteristics of children and the incidence of nutritional deficiencies in different parts of the country. It is our intention, therefore, to deal with some important aspects under the following heads: (a) growth in childhood and adolescence and (b) the incidence of nutritional deficiency.

## GROWTH IN CHILDHOOD AND ADOLESCENCE

**Height, Weight and Age:** Since much of the work has been done on school children the data for height, weight and age for a narrow age range only are available. In most surveys the recorded observations commence with the age group of five years and conclude with that of sixteen years, but at both ends of the scale the number of children is not large and hence it becomes difficult to find a suitable basis for comparison of the figures reported from different parts of India.

The observations on heights and weights of boys and girls are spread over the different provinces as follows:

STATE		Boys	Girls
Madras	...	1,858	1,548
Hyderabad	{ Telangana	...	8,379
	{ Marathwada	...	5,054
Orissa	...	1,167	—
Bengal	...	331	385
Assam	...	836	755
Bihar	...	1,563	360
United Provinces	...	969	—
Delhi	...	1,310	—
Punjab	...	1,591	—
TOTAL		23,058	3,048

It should strike even a casual observer that the data are pitifully inadequate. Figures for girls are almost negligible. Even for boys where larger number of observations are at hand the samples are so uneven in proportion to the general population and in each single instance are derived from such restricted localities within each region that one can

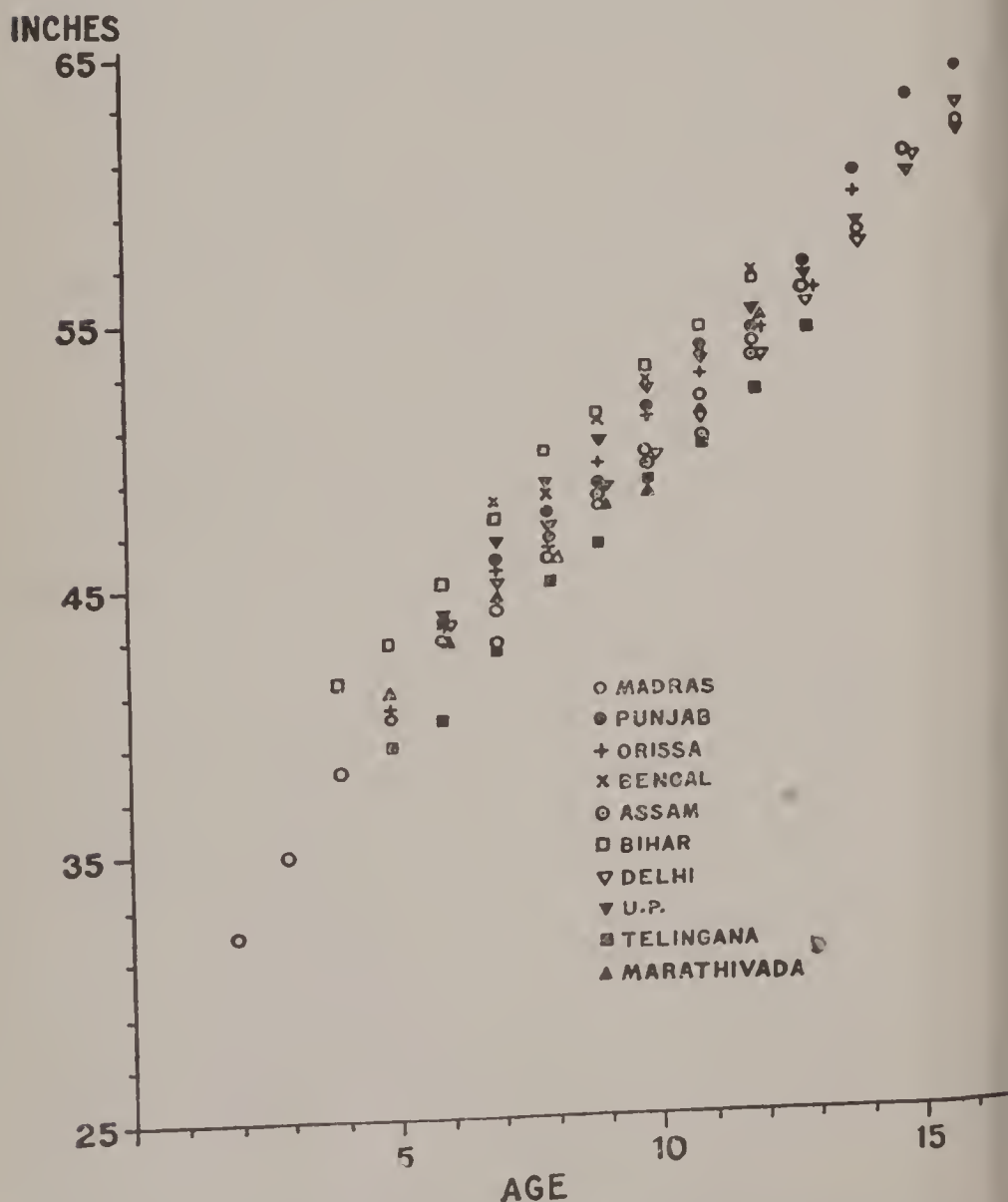


Figure IX—Heights of boys aged 2 to 16 years from different regions of India.

hardly consider them representative of the region. The heights and weights according to age are plotted in Figures IX and X. From the charts it becomes

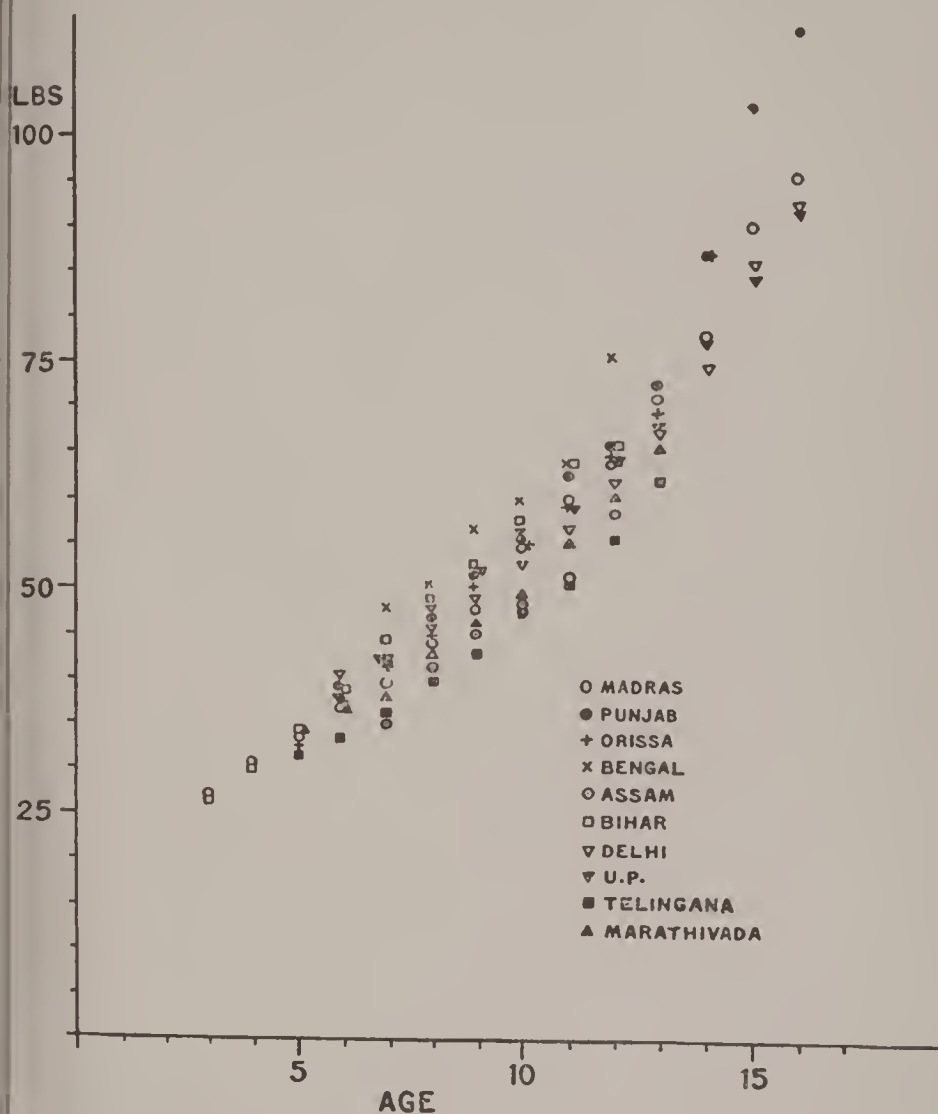


Figure X—Weights of boys aged 3 to 16 years from different regions of India.

clear that at no age between five and fourteen years the weights of boys from any of the regions in India approach the English or American values reported by Abrahams and Widdowson (1940). On the other hand, the heights of the English and American children at



five and six years are close to the highest Indian figure recorded for children in Bihar. At later ages the increase in height in the former group is more rapid and diverges appreciably from the height of Indian children. The height-weight charts also show that among Indian children there is a large spread of values at each age, the maximum difference in weights may be up to 15 lbs. and in height up to 7". Further, it does not appear as if there was a definite order of increasing height and weight for each age in which children from different Indian regions could be arranged. On the whole, however, it can be said that Bihar and Punjab are nearer the upper end of the region whereas Telangana (Nizam), Madras and Assam are near the lower end. Most of them, however, occupy different relative positions in different age groups. In addition to the data presented above, there exist others collected by Wilson and Widdowson (1942) in Punjab (5,969 children), and Central Provinces and Orissa (2,599 children). The former represented a wheat eating region and the latter a rice consuming area of India. The authors report that among the Punjabis, Hindu children tend to be lighter and shorter than Muslim and Sikh children of the same age. The authors believe that the differences may be racial and not dietetic. This may be partly true although not strictly so. The population in the north of India is derived from the various tribes and peoples migrating to India by the north-western route beginning with Aryans to the latest invaders from the Muslim countries, from Turkey down to Afghanistan. Consequently, there has been a mixture of all these racial groups and it is indeed a bold person who would attribute the existence of different racial characteristics

among the Indian people belonging to different religions in India. Further, the comparative figures

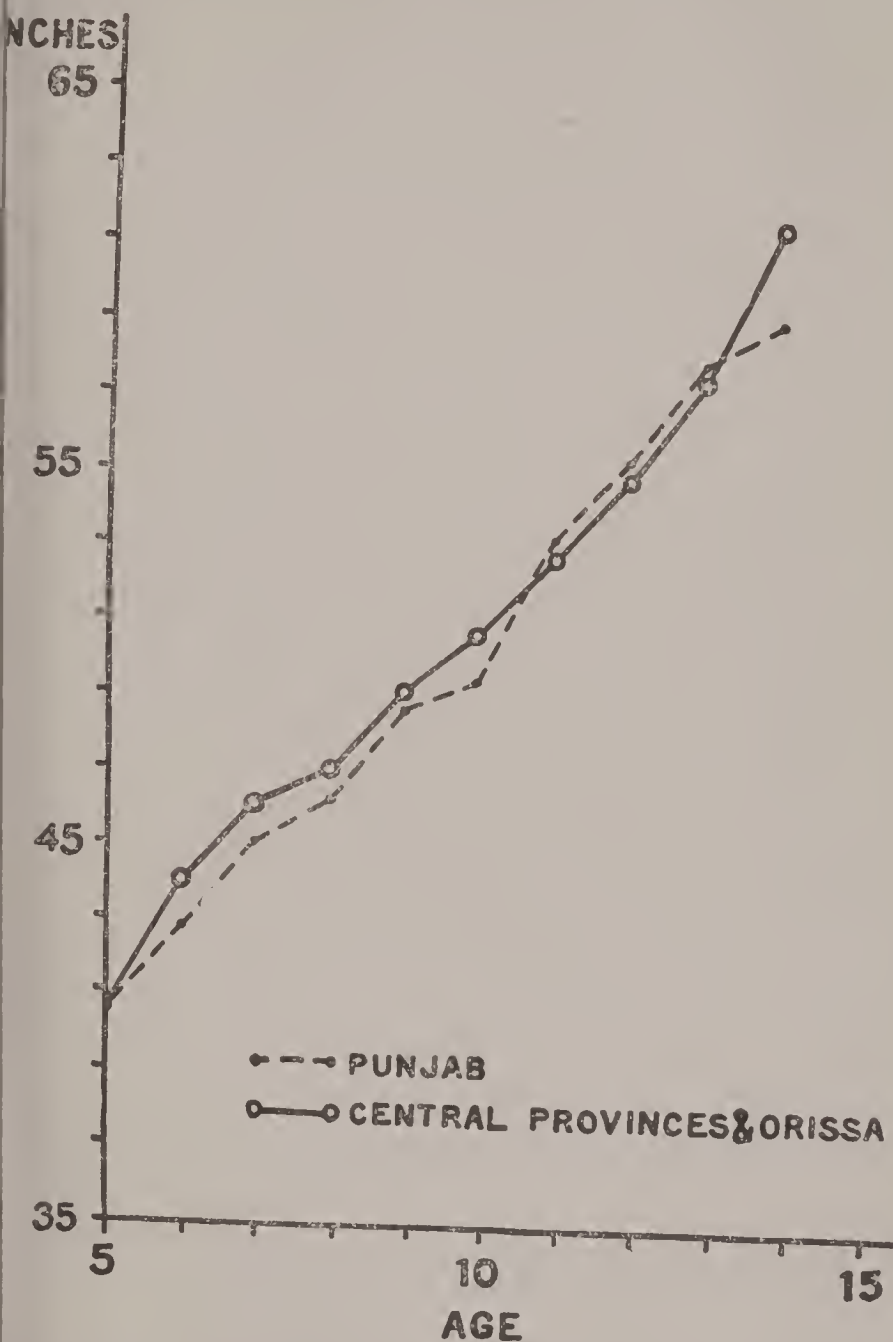


Figure XI—Heights of Hindu boys from wheat area of Punjab compared with the heights of Hindu boys from rice regions of Madhya Pradesh (Central Provinces) and Orissa.

given by Wilson and Widdowson for wheat eating Hindus of Punjab and rice eating Hindus of Central Provinces and Orissa are so close that they hardly

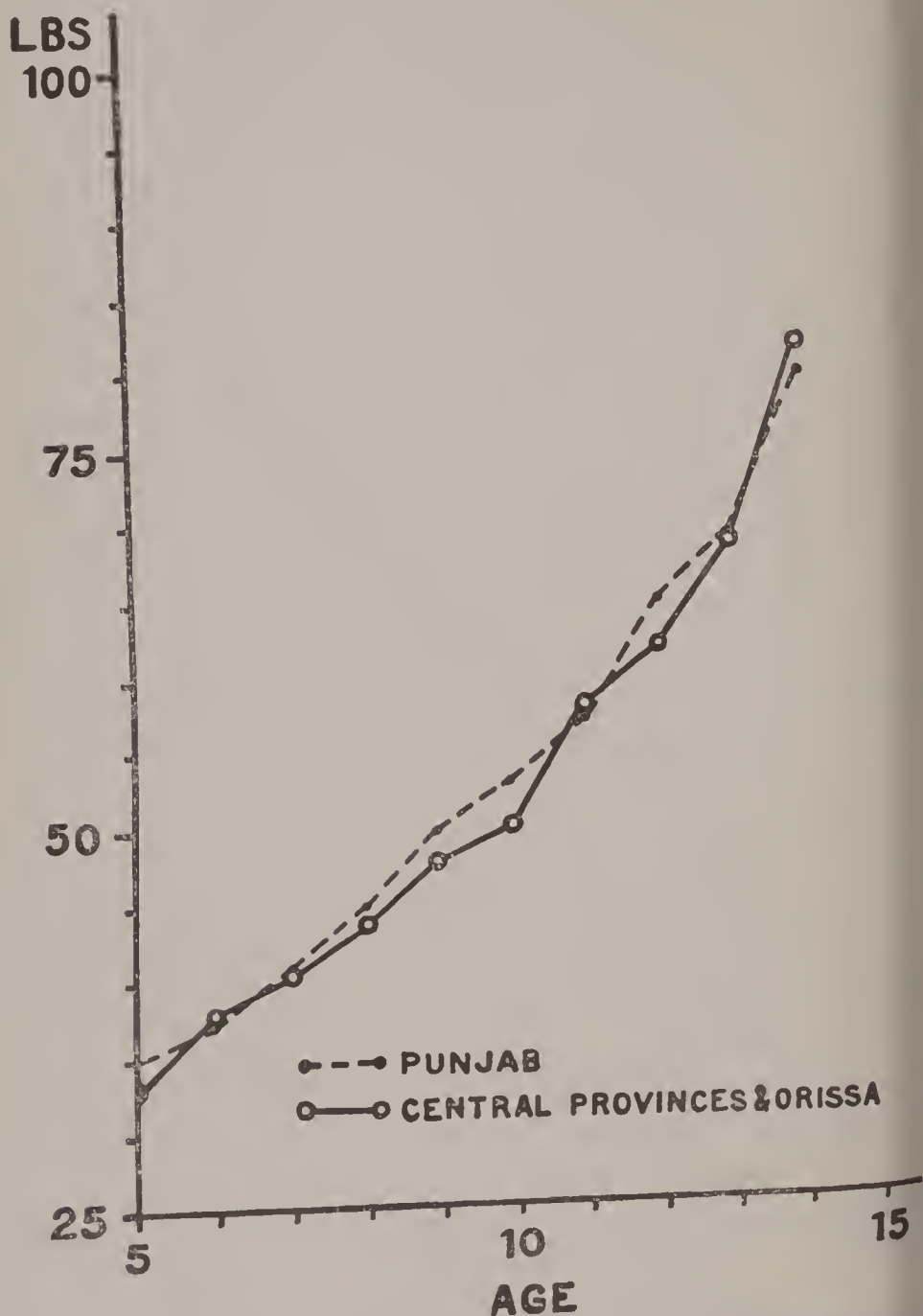


Figure XII—Weights of Hindu boys from wheat area of Punjab compared with the weights of Hindu boys from rice regions of Madhya Pradesh (Central Provinces) and Orissa.

support the racial hypothesis advocated by these authors to explain the height and weight differences. The actual growth curves for these two groups are given in Figures XI and XII. There appears to be hardly any significant and consistent difference in the heights and weights of boys from Punjab and Madhya Pradesh (Central Provinces) and Orissa.

From the data presented till now it is extremely difficult to make any generalisations regarding the differences in heights and weights of children from different Indian regions. A tentative conclusion may, however, be recorded that children in the north appear to be taller and heavier than children from the peninsular region. It is necessary to collect much larger body of data before the above conclusion can be considered to be fully proved. Among the factors which would determine these differences must be considered race and heredity and diets. It is not known to what extent one or all of these are operative. It must not, however, be concluded that the low height and weight are indicative of inhibited growth. This may be true to a certain extent but not wholly so. If malnutrition played a significant part in one part of the country and not in another, the effect should become manifest when weights are compared against heights for similar age groups. Such a comparison is illustrated in Figure XIII, from which it will become clear that weights cluster in a narrow region along a curve passing through largest number of points. For comparison the weights against heights are similarly plotted for English and American boys, in whom the weight for any given height is distinctly higher indicating beyond doubt that the English and American boys are heavier for the same height than the Indian boys. Here it may be reasonable to conclude that the

better type of dietary in England and America is probably responsible for the higher weights, but so far as the different regions in India are concerned the factors influencing weight in relation to height operate in the same way whatever they may be.

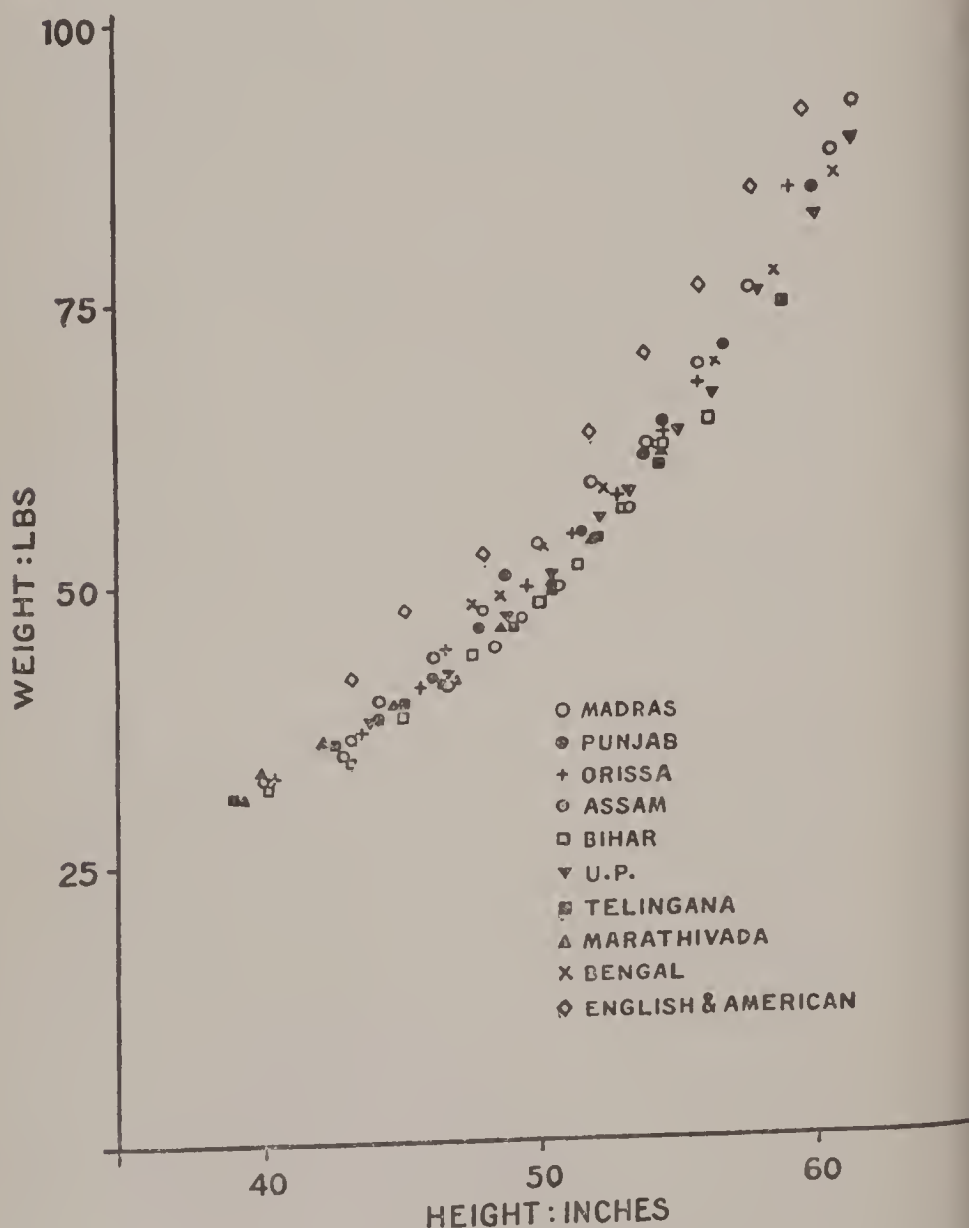


Figure XIII—Weight in relation to height of boys from different regions of India.



**Skeletal growth:** Apart from the records of heights and weights of children of school age, referred to above, there are very few observations on the normal growth of Indian children. Some studies of limited significance are available on the union of epiphyses in boys and girls. Hepworth (1929) examined 63 children in Ferozepore, Punjab, and concluded that in Indian boys and girls the union of epiphyses of the long bones took place  $2\frac{1}{2}$  to 3 years earlier than in English and American children. Galstaun (1930) examined 101 girls in Bengal and placed the age of union 3 to 4 years earlier than in European girls. Lall and Townsend (1939) examined 125 girls at Lucknow (Uttar Pradesh) and gave the following ages for the union of epiphyses:

		Age at Union Years
Humerus, lower end	...	14-15
Me dial epicondyle	...	15
Radius, upper end	...	16
Ulna, upper end	...	15
Radius, lower end	...	19
Ulna, lower end	...	19

Ledger and Wasson (1941) examined 189 boys and 49 girls of proved age in the North-West Frontier Province. They observed that in girls the union of epiphyses took place earlier than in boys and that in both, the age of union was lower than that obtaining in European and American children.

**Teeth:** Shourie (1946) has compared the eruption age of teeth in Indian and American boys. His observations in India cover 1,412 boys and 474 girls in Madras and 1,713 boys in Lahore between 5 to 21 years. He does not find significant differences in the

eruption age between these two groups. The combined data have been compared with the American figures and are given in Table XVII.

TABLE XVII—ERUPTION AGE OF TEETH IN INDIA

TOOTH		Eruption age in years	
		Indian	American
Central Incisor	...	7·10	7·34
Lateral Incisor	...	7·88	8·38
Canine	...	10·87	11·42
1st Premolar	...	10·50	10·21
2nd Premolar	...	11·57	11·00
1st Molar	...	6·48	6·59
2nd Molar	...	11·79	12·55

Unless some more data become available it is difficult to evaluate the significance of the small differences to be noticed between the ages in columns 2 and 3.

The above discussion should make it clear that plenty of information on different aspects of child growth is needed before any idea of normal growth in Indian children can be obtained.

### NUTRITIONAL STATUS OF CHILDREN

In the early days the only method of determination of nutritional status used all over the world was clinical, based on the considerations of height and weight in relation to age, muscle tone, carriage and gait, subcutaneous fat, and the colour of mucous membranes. The interpretation of the results of such clinical examination is extremely difficult and has been

criticized from time to time by various experienced workers among whom may be mentioned Kruse (1943), Dann and Darby (1945) and Sinclair (1948). In the absence of any specific criteria by which to judge the deviation from normal nutriture, the clinicians used to record their impressions in terms of good, fair and poor. Later studies revealed that certain clinical signs and symptoms could be specifically ascribed to deficiencies of particular nutrients. The records of manifest signs and symptoms of deficiency states began to be included in nutrition survey schedules. Conditions like xerosis of conjunctiva, Bitot's spots, xeroderma, phrynoderma, angular stomatitis, red raw or pale glazed atrophic tongue, stigmata of past rickets, etc. were a few which received special attention.

Apart from these, several anthropometric measurements were usually taken and indices calculated to yield some information on nutriture. The authors above referred to have discussed in detail the inadequacy of anthropometric indices for such work. We find it unnecessary to go over the same ground.

The technique of nutrition surveys as used in India has undergone changes in parallel with those observed elsewhere. There were periods when mere clinical classification into good, fair and poor held sway. This was followed by the adoption of anthropometry as an aid to assessment of nutriture. Anthropometric studies in their turn have been superseded by methods in which greater stress is laid on deficiency signs and symptoms, the physical measurements being reduced to the bare minima of heights and weights.

Still more recently, biophysical and biochemical methods of assessment have been brought into use,

but are restricted to certain well equipped laboratories only. The routine nutrition surveys in India have mainly depended upon clinical appraisal of the general condition together with the recording of signs and symptoms of nutritional deficiencies. The validity of the latter as a means of assessing nutriture has been rightly questioned by Sinclair (1948). The Committee on Nutrition of the British Medical Association (1950) has also taken the same view. Although it is true that the early clinical manifestations like folliculosis, angular stomatitis, changes in gums, etc., are likely to be due to more than one deficiency or even due to non-nutritional causes, it is not less true that some of these conditions in India are so typical and have so often been proved by therapeutic tests as being specific nutritional deficiencies, that one may be justified to a certain extent in including them as evidences of the latter.

It is extremely difficult to judge the nutritional significance of the clinical classification of children into good, fair and poor without the knowledge of criteria used by the various investigators. Certain workers in India tried to correlate their clinical impressions with anthropometric indices. Aykroyd, Madhava and Rajagopal (1938) tested Franzen and Palmer's (1934) A.C.H. index on 4,136 boys and 510 girls in South India. They found that in a large number of children the sum of the arm girth, measured flexed and extended, was smaller than the sum of anteroposterior diameters of expanded and contracted chest; besides, the intertrochanteric distance in 50 per cent of the children examined was smaller than 20 cm. The authors conclude that if provision is made for lower hip width and lower values of G, the modified A.C.H. index could be applied to South

Indian children between 6 and 13 years. Wilson, Ahmad and Mitra (1937) examined 3,000 boys in Calcutta and 1,250 in Ferozepore, Punjab. In Calcutta, they found poor correlation between the "selected" children and the malnourished as determined clinically. Although in Punjab the results were more encouraging, the authors do not seem to be altogether satisfied with the utility of the index. They, however, recommend further work on the problem. Some other authors have also used the A.C.H. index, and although all of them appear doubtful about its utility none except Aykroyd *et al* (loc. cit) has suggested any modifications. The experience of these other authors confirmed the earlier findings that in Indian children as a whole there was little correlation between "selection" by A.C.H. index and the occurrence of malnutrition as judged by gross clinical manifestation. It is not surprising that the index has fallen into disuse so far as the nutrition survey work in India is concerned.

Mitra (1941 and 1942) examined approximately four thousand children to determine their nutriture by Knudsen-Schiøtz index (Bigwood, 1937) and compared the results with those obtained by clinical examination. For the purposes of the index the continuity and straightness of the dorsal median furrow is examined, for it is believed that in malnourished children the furrow is broken and is unduly sinuous. In 1,521 children in Jharia coal fields, Mitra observed that the index selected a larger number of children as malnourished than could be determined by clinical examination. In another batch of 2,000 children in Singhbhum district he obtained quite the opposite results. Thus the application of this index did not carry the solution of the problem



of determining nutriture by anthropometry any further.

Kurulkar (1948) has recently examined the problem from another angle; he has tried to correlate the foot length, pelvic width and stature in an erect human body in an attempt to find a rough measure of volume for a normal body. His index called the C index is based on the following formula:

$$\frac{2 \times \text{Weight in grammes}}{\text{Stature in cm.} \times \text{Foot in cm. length} \times \text{Pelvic width in cm.}} = C$$

In short it was an attempt to correlate volume with body weight. The values of C varied from 0.7 to 1.54 and showed a good fit between 4 to 22 years with a parabolic curve of the formula  $Y = 0.45 \times X^{1.6}$  where  $Y = C$  index and  $X$  age in years. Kurulkar's observations cover 5,789 individuals between 4 and 25 years. The C index of individuals clinically graded poor (independently by two medical examiners) was below that of the normals. Kurulkar further develops the idea of determining a myotrophic index which is obtained by applying age and adiposity correction to the C index. Adiposity is determined by measuring the thickness of the skin of the anterior axillary fold. The conception is attractive and should appeal to those who believe in the application of somatometric indices to determine nutriture.

In our opinion, the surveys which have recorded certain signs and symptoms of deficiency states have given more information on the prevalence of malnutrition in India than the anthropometric studies. Bitot's spots, phrynoderma and angular stomatitis have been recorded in most surveys carried out between 1937 and 1942 and hence a comparison of the incidence of these conditions is instructive. In

Figure XIV is shown the comparative incidence of these conditions. It would be hazardous to generalise, but the fact does strike one that as one proceeds from south to north the incidence of Bitot's spots decreases,

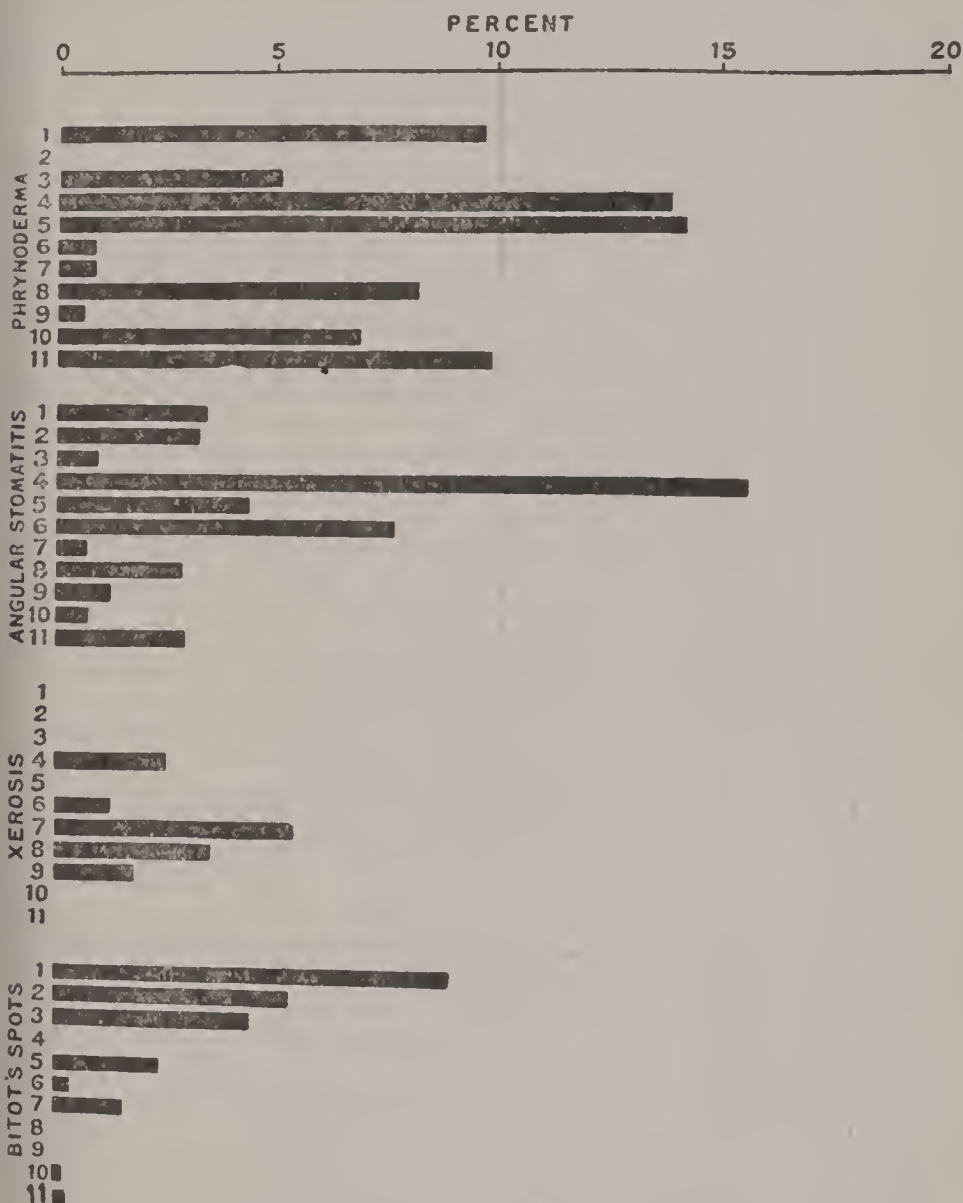


Figure XIV— Incidence of certain nutritional deficiency conditions in India.  
 1. Travancore. 2. Hyderabad. 3. Bombay. 4. Orissa.  
 5. Central Provinces. 6. Bengal. 7. Assam. 8. Bihar.  
 9. Delhi. 10. Punjab. 11. Kashmir.

it is the highest in Travancore and lowest in Punjab. If there were available proofs from the diet surveys that deficiency of vitamin A in diets progressively decreased from south to north, one could find an explanation for these observations. The dietary habits and the comparatively larger consumption of milk and milk products in the north would suggest such a possibility and this aspect is well worth further exploration.

From the year 1943 onwards the food situation in India has progressively worsened and its effect has been seen in the dietaries of the people. Although there has not been much difference in the consumption of cereals, that of protective foodstuffs has materially suffered and it is but inevitable that malnutrition should have been on the increase. Records of nutrition surveys for the post-war period on an extensive scale are not yet available for comparison, a few examples may, however, be referred to. Table XVIII includes the information gathered from repeat surveys in certain localities.

TABLE XVIII—THE INCIDENCE OF DEFICIENCY STATES AS FOUND IN REPEAT SURVEYS

Locality and Year .		Number Surveyed	Phryno-derma %	Angular Stomatitis %	Bitot's Spots %
COONOOR *					
1937	...	777	8.6	8.7	2.1
1949	...	566	20.0	1.0	3.4
ORISSA †					Xerosis %
1942	...	11,601	14.6	16.5	2.7
1945	...	7,711	4.1	14.6	2.7
1947	...	700	8.0	7.0	7.8

\* Nutrition Research Laboratories—Unpublished Data.

† Orissa Government—Department of Public Health Report, 1947.

The picture presented by the above data is not only confusing but probably misleading owing to the fact that the nutrition surveys to date have laid too much stress on recording only a few manifestations of malnutrition excluding others and the information obtained from diet surveys does not necessarily indicate why the shift in incidence of the three conditions mentioned above should have taken place.

The Indian nutrition workers as a whole were not satisfied with the methods according to which the nutrition surveys were being carried out. In 1948, the Nutrition Advisory Committee of the Indian Research Fund Association considered the problem afresh and formulated three schedules for the surveys. These were intended for (1) Rapid Nutritional Surveys, (2) Routine Nutritional Surveys and (3) Nutrition Surveys supported by laboratory investigations. These schedules are still on trial and it is too early to say if they would prove satisfactory. For details about these schedules the reader may consult the published reports of the 17th and 18th meetings of the Nutrition Advisory Committee.

In Europe and America, finer techniques such as biomicroscopical, biophysical and biochemical methods are being evolved. The work of Kruse, on the one hand, and Bessey and Lowry on the other has added a great deal to our knowledge of morphological and biochemical changes in early deficiency states although the significance of the former has been seriously questioned and that of the latter can only be judged by further work. One practical consideration has prevented their application in India to nutrition studies. Apart from the controversy raging around Kruse's interpretation of biomicroscopic findings, the fact that in any part of India there is always

an appreciable proportion of child population showing manifest signs of deficiency has made it unnecessary to resort to finer techniques. The deficiency conditions literally stare in one's face in any survey on school children and hence it becomes a luxury to utilise more elaborate and time consuming methods than a careful clinical appraisal.

The time is yet far off when the clinical appraisal will fail to give information about the extent of malnutrition making it necessary for workers in India to resort to laboratory methods. This is not to decry the usefulness of the newer methods. It should be understood that our problem at present is not so much the diagnosis of latent and potential deficiency states as it is to prevent manifest deficiency disease from occurring as widely as it does.

In spite of these considerations some workers in India have studied early deficiency states and their work on dark adaptation and vitamin A status, saturation and dermal tests for vitamin C nutrition, excretion of thiamine and riboflavin in deficiency states, etc., should bear testimony to the fact that latent and potential deficiency conditions are not being altogether neglected.

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## CHAPTER XVI

### BLOOD

TILL RECENTLY there was very little information on normal haematological standards for the Indian population. Although there was little reason to believe that in the normal healthy Indians the values would materially differ from those obtained by workers in Americans and Europeans, it was desirable to have figures available for the Indian population. Some workers in India realised the importance of this subject and undertook studies to establish the normal range of values. Among them must be mentioned Napier and his school at Calcutta who did much to establish standards in Bengal by using reliable methods. Sokhey and his colleagues also collected some valuable information in Bombay. Later studies in other parts of India showed that there was little difference in the average red cell, haemoglobin, packed cell volume, and total and differential leucocyte counts obtained in the normal healthy Indians of comparable age groups in any part of India, provided of course, the environmental conditions were such as not to affect any of these values materially. In Table XIX are given the results of several such studies. Haematological constants in the table have been calculated from the data in a few cases where the authors have not provided them.

The table reveals some minor differences in the different values. For both men and women there seems to be greater agreement in the figures for red blood cells than for haemoglobin content of blood. For men there is divergence even in the figures

TABLE XIX—HAEMATOLOGICAL STUDIES ON ADULTS IN INDIA

Author and Year	Locality	Sub. No.	R.B.C. mill. c.mm.	Haemo- globin gm. %	P.C.V. %	M.C.V. cu $\mu$	M.C.H. $\gamma$	M.C. H.C. %	W. B. C. Total/ c.mm.
M E N									
Napier & Das Gupta	Calcutta	50	5.362	14.77					6,542
Do	"	30	5.533	15.70	50.53	90.49	28.53	31.07	—
Sokhey <i>et al</i> *	Bombay	121	5.11	15.37	41.72 (44.14 corr.)	86.38	30.08	34.82	—
Prasad & Chowdhury	Patna	37	—	14.38	—	—	—	—	—
Khanna & Sachdeo	Lahore	75	5.17	14.78	47.0	90.7	28.44	31.44	—
Gokhale & Lokre	Indore	25	5.354	15.35	—	—	—	—	7,248
Ramalingaswami & Venkatachalam	Coonoor & Ootacamund	100	5.36	15.86	49.27	92.27	29.69	32.25	7,288
W O M E N									
Napier <i>et al</i>	Calcutta	128	4.615	12.58	—	86.82	27.42	31.57	7,162
Sokhey <i>et al</i> *	Bombay	101	4.465	12.99	36.27 (38.37 corr.)	85.94	29.10	33.86	—
Prasad & Chowdhury	Patna	29	—	13.79	—	—	—	—	—
Khanna & Sachdeo	Lahore	25	4.612	13.06	41.8	90.6	28.17	31.45	—
Gokhale & Lokre	Indore	25	4.612	13.70	—	—	—	—	8,076
Venkatachalam	Coonoor	17	4.96	12.76	42.5	85.80	25.41	30.04	7,782

\* Constants have been calculated after correcting the P.C.V. for shrinkage. The corrected value for P.C.V. is given within brackets.

obtained by Napier *et al* in two series of observations. The explanation which these authors give is that in their first series of 50 there were included 16 individuals of lowest income groups (probably malnourished) and the values obtained with them may have lowered the average of the whole series. It would have been easy for them to find out by reference to individual figures whether it was actually so. Since that was not done it is difficult to assess how far their argument is valid. Prasad and Chowdhury (1943) who used a similar technique in Bihar found for 37 "well-to-do" adults an average value of 14.38 gm. per 100 c.c. of blood and Khanna and Sachdeo (1946) reported from Lahore only slightly higher average of 14.78 gm. It may, however, be assumed that in general, the normal haemoglobin content of Indian males appears to be 15 gm. per cent or slightly over. The highest values for haemoglobin have been reported by Ramalingaswami and Venkatachalam (1950) for their subjects from Coonoor and Ootacamund. These towns are situated at an altitude range of 6,000-7,500 feet above sea level and this may be the underlying reason for the higher values of haemoglobin. While commenting on them in relation to Wintrobe's absolute constants for blood the authors suggest that the haematological response to altitude which acts like a chronic anoxic stimulus does not primarily involve an increase in circulating erythrocytes and haemoglobin but that an increase in the corpuscular surface area and hence a close correlation between cell volume, cell number and cell haemoglobin are of greater significance.

A much greater divergence of values is, however, seen in the reported figures for packed cell volume. It is probable that much of this difference can be

explained on the basis of different techniques adopted and on the differences in the average red blood cell count. That it is not all can be seen by reference to a critical study reported by Sokhey *et al* (1937). They refer to the observations on shrinkage caused by the use of potassium oxalate as an anti-coagulant. Osgood (1926) found in two samples of blood a shrinkage of 3.75 per cent as against the determination made on blood where heparin was used as the isotonic anti-coagulant. Wintrobe (1929, 1931 and 1932) who also used heparin and potassium oxalate gives three different figures for shrinkage 3.7 per cent, 5.75 per cent and 8.2 per cent, and without giving any reasons for such divergent figures used the last value in making allowance for shrinkage in the calculation of cell volume. Haden (1929-1930) using hirudin for comparison finds a shrinkage of 8.0 and 8.24 per cent. Napier and Das Gupta (1936) compared shrinkage due to potassium oxalate against hirudin and heparin and reported values of 6.86 per cent and 9.19 per cent respectively. If it is assumed that isotonic heparin and hirudin do not cause shrinkage of cells, the results obtained by comparison with these anticoagulants should give the same value for corpuscle shrinkage within the limits of experimental error. Moreover, there is little apparent reason for the large variations in shrinkage reported by the same author and by different authors. Sokhey *et al* (1937) kept the conditions regarding the concentration of isotonic anticoagulants and of potassium oxalate constant and varied the speed of centrifugation. They found, as was to be expected, that at lower speeds of centrifugation the time for obtaining constant packed cell volume lengthened. But when full packing was ensured their results with different reagents used as



anticoagulants were within the limits of experimental error. The shrinkage due to potassium oxalate was 5.78 per cent as compared against hirudin and 5.89 per cent against heparin, thus proving the contention that ideal conditions for determining cell volume did not exist in many of the studies referred to above. It may be reasonable to conclude, therefore, that there was little justification for Napier and Das Gupta to use an arbitrary factor of 1.09 for correcting the figure for packed cell volume obtained by the use of potassium oxalate. Ramalingaswami and Venkatachalam (1950) used a mixture of ammonium and potassium oxalates which, according to Wintrobe (1946), causes no shrinkage, hence there was no need to apply a shrinkage correction. The high cell volume reported by them may be due partly to the rather high erythrocyte content and partly to the response to chronic anoxic stimulus resulting from the high altitude at which their subjects were living.

There are few observations on the blood of children. One such study is by Napier and Das Gupta (1940) who found that at five years both boys and girls had 11.5 gm. haemoglobin per 100 c.c. blood. Thereafter it showed a steady increase, the increase for girls became slower at 11 years; at 16 years the haemoglobin values for boys and girls being 13.5 gm. and 12.8 gm. respectively. The authors did not find any differences between Bengalee and Sikh boys with respect to the haemoglobin levels.

The mean red cell diameter has come in for study by several investigators. Sankaran and Rao (1938) used the method of Hynes and Martin (1936) as modified by themselves and have reported figures for South Indian men and women. Napier, Sankaran, Swaroop and Rao (1939) and Napier, Sen Gupta and

Chandrasekar (1941) applied the above method for the study of mean red cell diameter in Bengalees.

The comparative figures are given below:

### MEN

	Subjects	Mean r. b. c. diameter $\mu$	Standard deviation
Sankaran and Rao (1938) ...	15	6.85	—
Napier <i>et al</i> (1939) ...	50	7.288	—
Ditto (1941) ...	25	7.3438	0.1328
Rao and Rao (1942) ...	25	7.079	0.139

There is a discrepancy between the two sets of observations reported by Napier *et al*. The authors themselves are of the opinion that their later figures are correct since they point out certain difficulties and defects in technique which may have affected their earlier results. On the other hand the values reported by Sankaran and Rao, and Rao and Rao, also do not agree although one of the authors had participated in both these investigations. Rao and Rao state that the lower red cell diameter observed by Sankaran and Rao was in the inhabitants of Coonoor, a place which is at an altitude of 6,000 feet, which must have been responsible for reduction in red cell diameter, whereas their own subjects were from the plains. In support of this conclusion, Rao and Rao quote their findings in 25 British European subjects from Wellington (6,000 feet above sea level) in whom they found a mean red cell diameter of  $7.135 (\pm 0.130) \mu$  a figure which is lower than the average 7.202 given by Price Jones (1933). They also quote Gulland and Goodall (1914) to the effect

that "the size shows very little variation in health. The average size, however, is considerably reduced in the case of the inhabitants of high altitudes." Later work, however, lends little support to this hypothesis. On the other hand it indicates an increase in the surface area of the corpuscle in persons residing at high altitudes. Although the importance of red cell diameter in physiological studies has decreased with the acceptance of absolute mean corpuscular volume in blood (Wintrobe 1946), it is somewhat disconcerting to find that no satisfactory explanation can be found for the observed discrepancies in results reported by different authors in spite of the fact that identical procedure was used in these investigations.

### PLASMA PROTEINS

Considering the importance of plasma proteins it is astonishing how little attention had been paid to their determination in healthy Indians. Lloyd and Paul (1928) give a figure of 8.152 gm. (average of 8 subjects) and 7.522 gm. (average of 10) per 100 c.c. serum. They used Robertson's refractometric technique. Later Mudaliyar, Sundaram and Ramachandran (1933), using the colorimetric technique of Wu, found an average of 6.135 gm. for 9 subjects. These authors further fractionated the serum proteins by precipitation with different concentrations of  $(\text{NH}_4)_2\text{SO}_4$  and determined the protein fractions colorimetrically. Chopra, Mukherjee and Sunder Rao (1934) also utilised the refractometric technique and reported for 11 healthy subjects the following average values; total protein 7.44 gm., albumin 4.61 gm., globulin 2.83 gm., pseudoglobulin 2.65 gm., and euglobulin 0.18 gm. In 1947, Datta and Chakravarty published the results of an investigation on

healthy men and women. These authors also have used Howe's method for fractionating plasma proteins, but all their protein estimations were carried out according to microkjeldahl procedure which is free from sources of errors inherent in the methods used by the previous investigators. Their results are summarised in Table XX.

TABLE XX—PLASMA PROTEINS IN INDIAN ADULTS

	Men			Women		
	No.	Mean gm./100 c.c.	St. deviation	No.	Mean gm./100 c.c.	St. deviation
Total protein ...	45	7.51	0.23	25	7.49	0.27
Albumin ...	45	4.93	0.20	25	4.87	0.21
Globulin ...	45	2.58	0.16	25	2.60	0.18
Euglobulin ...	35	0.52	0.10	25	0.58	0.12
Pseudoglobulin ...	35	1.84	0.17	25	1.74	0.21
Fibrinogen ...	35	0.25	0.09	25	0.26	0.09

Gokhale and Chitre (1950) utilising similar technique found the following average values for 40 healthy adults. Total serum protein 6.93 gm., Albumin 4.70 gm., Globulin 2.26 gm., with albumin: globulin=2.08. The albumin/globulin ratio calculated from Datta and Chakravarty's figures (making allowance for the fibrinogen content) amounts to 2.21.

The latter authors have compared their values with those reported by several authors abroad. It appears from these figures that there is little difference between the plasma protein content of Indian and European subjects. Datta and his colleagues have also studied the effect of certain pathological conditions on

TABLE XXI—NITROGENOUS AND  
OTHER CONSTITUENTS OF BLOOD IN INDIANS

Particulars	No. Subjects	Range mg.	Mean mg.	Reference	Remarks
<b>BLOOD</b>					
Non-protein N.	191	17—36·0	25·6	1, 2.	Laked blood Values averaged.
„	126	15·17—21·92	18·43	3.	Unlaked blood.
Urea N	317	5·6—18·0	11·56	1,2,3.	Values averaged.
Amino acid N.	126	2·71—4·98	3·88	3.	
Uric Acid	186	1·98—3·82	2·70	1, 3.	Values averaged.
Creatinine	257	0·73—2·70	1·52	2, 3.	Do.
Sugar	60	80—120	100	1	
Cholesterol	60	91—150	119·3	1	
„	131	100—250	159	2	
„	104	130—184	146	4	
„	100	82—184	116	5	
<b>SERUM</b>					
Calcium	10	—	10·54	6	
	16	11·91—13·08	12·51	7	
	60	8·9 —10·2	9·60	1	
Inorganic Phosphorus.	10	—	2·82	6	
	16	2·91— 5·05	3·76	7	

- |   |                         |                    |
|---|-------------------------|--------------------|
| 1. Bose (1927).                         | 2. Chakrabarty (1948).  | 3. Gokhale (1939). |
| 4. Ghose (1933).                        | 5. Boyd and Roy (1927). | 6. Roy (1942).     |
| 7. Hughes, Srivastava and Sahai (1929). |                         |                    |



plasma protein levels; some of their findings will be referred to in another chapter.

**Other Constituents:** The available information about certain other constituents of the blood is given in Table XXI. Data for major nitrogenous constituents, sugar, cholesterol, calcium and inorganic phosphorus are included in the table. Where the range of variation and the average values obtained by different authors differed widely, separate values have been given; where the differences were small the values have been averaged. No figures are available for several other blood constituents apart from some vitamins which have been referred to in other chapters.

It will be clear from the information given in this chapter that the blood of healthy Indians does not on the whole differ in its composition from that of human beings of different ethnological stock, living under vastly different environment and with dietary habits basically different from those prevailing in India. This point needs to be stressed on account of marked differences in the basal energy metabolism and the level of nitrogen in urine to both of which reference has already been made. On the other hand the information given above provides additional evidence, if any were needed, in support of the concept of constancy of the composition of Claude Bernard's *milieu interieur*.

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## CHAPTER XVII

### ANAEMIA

IN INDIA anaemia of varying degrees and of varying types seems to be fairly widespread among the poorer classes of the population. The underlying reasons for this state of affairs are probably manifold. A diet which is marginal with regard to quality proteins, minerals and other accessory food factors can hardly be conducive to the maintenance of haematological levels at the normal under stresses and strains inseparable from life under ordinary circumstances. Tropical diseases resulting from bacterial infections and parasitic infestation are active in creating conditions favourable for anaemias to develop. Certain other diseases may be the direct cause of anaemia. As an illustration may be cited, a recent report by Aiengar *et al* (1947) about the conditions in the malarious district of Malnad of Mysore State. They state that on an average about 500 cases of anaemia are admitted annually to the Shimoga Hospital. According to them, malnutrition coupled with malaria and hookworm disease cause such a high incidence of anaemia. In another area in South India, Ramalingaswami and Patwardhan (1949) found in a sample labour population on tea, coffee and rubber plantations a high incidence of anaemia. Out of 338 persons examined 291 had subnormal haemoglobin levels and 148 of these had less than 10 gm. per 100 c.c. Almost similar conditions have existed in tea plantations in Assam. Napier and Das Gupta (1937, 1938) found the tea garden labourers of both sexes generally suffering from microcytic hypochromic

anaemia of iron deficiency type. Out of a sample of 100 individuals in one locality, 86 were found infected with hookworm. The authors feel that the above infection together with malaria and defective diets were the causative factors. Macdonald (1939) seems to be of the same opinion. Hare (1940) believes that conditions in Assam were not necessarily such as to increase the anaemic state of the labourers. He found anaemia among newly recruited labour which came from outside the province of Assam and suggested that it only represents a condition common among the poor of India probably arising out of a long-standing defect in diet. Hare's contention is largely borne out by the experience with army recruits during the last war. Hynes, Ishaq and Morris (1945) found that in a batch of 600 recruits only 195 had haemoglobin levels of 15 gm. per 100 c.c. or above. In a majority the anaemia was normocytic and normochromic and improved on service diet, but responded better if supplemented with iron medication. Hynes, Ishaq, Morris and Verma (1946) report on a further batch of 801 South Indian Army recruits with anaemia associated with poor muscle development and presence of ocular and skin conditions suggestive of nutritional deficiencies. In both the above investigations the authors point out the fact that although the evidence of hookworm infestation was present in a majority of recruits, there was little correlation between the hookworm load and the degree of anaemia. Hynes, Ishaq and Verma (1946) studied the effect of various diets on the anaemias in 800 army recruits. The diets fed consisted of the standard army ration supplemented with meat or milk and with or without ferrous sulphate 3 to 6 gr. a day. They confirmed the findings of Hynes, Ishaq and



Morris (*loc. cit*) that iron medication improved the response already obtained with the standard army ration. Extra meat or milk did not improve the performance seen on the standard army ration. They also found that hookworm infestation did not interfere with the haematological response to good diet whether it was supplemented with iron or not.

It is somewhat surprising that there are no observations on the status of the blood in nutrition surveys on school children. One can expect the effect of chronic subnutrition to be seen better among children than in adults. The incidence obtaining in adults leads one to suspect that children of the poorer classes might also be suffering from anaemia to a varying degree. It would be desirable for the nutrition workers in India to direct their attention to this important aspect of the problem.

**Nutritional Macrocytic Anæmia:** The description of the incidence of anaemia given till now referred to the normocytic, microcytic, normochromic or hypochromic varieties only. Several observations are on record to show that macrocytic anaemias are not uncommon in this country. Of these, the true pernicious or Addisonian anaemia is rare, on the other hand, nutritional (tropical) macrocytic anaemia (NMA) and macrocytic anaemia of pregnancy are fairly widespread. During his investigations on Assam tea plantation labourers, Napier has noted the presence of macrocytic anaemia. Wills (1934) had shown that NMA was common among both sexes. She had obtained good therapeutic results with marmite and had suggested that thiamine and riboflavin were not the active components. Wills had also noted that the factor present in yeast extract and to which NMA responded was inactive

in Addisonian anaemia. These observations led her to believe that NMA was a deficiency disease probably caused by a lack of Castle's extrinsic factor. Napier (1939) also expressed the opinion that it was a deficiency anaemia, but that it differed from the pernicious anaemia in that the deficient factor was different from the extrinsic factor of Castle. Taylor and Manchanda (1940) described 24 cases in adults of 24 to 60 years from the poorest classes in Punjab and were of the opinion that poor dietary was one of the aetiological factors. Ahmad (1944) reported 5 cases from the United Provinces, Das Gupta *et al* (1946 a and b) 23 cases from Calcutta and Sundaram (1944) 15 cases and Ramalingaswami and Menon (1949) 6 cases from Madras Province. Taylor and Chuttani (1945) found that whereas in meat-eaters in the army the anaemia was mostly normocytic normochromic, in vegetarians it tended to be macrocytic.

Thus it will be clear that NMA occurs all over India, its extent of incidence can hardly be judged by the limited number of cases referred to above. It is quite probable that only a small fraction of cases actually occurring go to the hospitals and that too when the anaemia has become sufficiently severe or when other complications have set in. The opinion of Wills that it may arise out of dietary deficiency has already been referred to.

Although Wills had found marmite active against NMA, the actual therapeutic trials excluded thiamine, riboflavin, nicotinic acid, pyridoxin, pantothenic acid, and p-aminobenzoic acid (Wills 1945, Moore *et al* 1944). Trials with refined and crude liver extracts gave contradictory results. Wills, Clutterbuck and Evans (1937) and Wills and Evans (1938) had found refined liver extracts ineffective in NMA.

Sundaram (1944) and Patel (1946) on the other hand found proved cases of NMA responding to refined liver extracts provided adequate doses were given. In the meantime the work of Spies and others had demonstrated the therapeutic effect of folic acid in Addisonian anaemia, NMA, macrocytic anaemias of pregnancy and of sprue. Although later work showed that folic acid was not a desirable therapeutic agent in Addisonian anaemia its value in other macrocytic anaemias has now been established and some recent trials in India by Das Gupta and Chatterjea (1946), Kemp (1947), Goodall *et al* (1948) and Ramalingaswami and Menon (1949) have confirmed these findings, if any further confirmation were actually needed. The last mentioned authors have given a probable explanation of the contradictory results obtained with refined liver extracts. While discussing the bone marrow morphology they point out that two types of marrow reactions may be seen in NMA, one which is megaloblastic resembling that seen in Addisonian anaemia, and the other which could be described as early normoblastic. The cases with megaloblastic reaction may respond to refined liver extract whereas those with normoblastic reaction are refractory. Both, however, respond to crude liver extract and folic acid. On the other hand, Bhende and Patel (1948) were of the opinion that activity or otherwise of refined liver extracts against NMA depended upon the uncertain and variable extent to which certain active factors were removed during the process of purification of the liver extracts. The explanation offered by Ramalingaswami and Menon appears to be more plausible in view of the support derived from the interpretation of the bone marrow findings in NMA cases.

**Macrocytic Anaemias of Pregnancy:** Balfour (1927) was the first to undertake a systematic study of this condition in India. She studied 150 cases in Bombay between 1925-1927, most of which were derived from the lower middle and labour classes. Balfour felt that malaria, dysentery, hookworm and sprue were not the aetiological factors. She, however, did not give any definite opinion regarding the role of poor diets. Wills and her colleagues later studied this problem more closely and have provided information of great value. Wills and Mehta (1929) found that the incidence of anaemia was spread over all the communities. The authors were struck by the fact that the largest number of cases were from the poor classes. This led to a study of the diets of different economic groups in Bombay. Wills and Talpade (1930) who made exhaustive study of the dietaries of middle and labour classes in Bombay City found that in the former the diets were adequate and cases of pregnancy anaemia few. On the other hand, the diets of labour class families were qualitatively and quantitatively inadequate. There were deficiencies of vitamins A and C, fat content was low and vitamins of the B complex were also inadequate. The authors, therefore, suggested that a deficiency of one or more of these factors predisposed women to anaemia which came into prominence when additional stress of pregnancy had to be borne. In 1933, Wills found that marmite was useful in treating the "pernicious" anaemia of pregnancy.

It was at one time suggested (Editorial, Ind. Med. Gaz. 1932) that the disease be called Tropical Anaemia of Pregnancy for the reason that the condition was extremely rare in temperate regions. According to the information then available, only ten cases of



macrocytic anaemia in pregnancy had been reported in Europe in a total of 105,400 pregnancies, i.e., an incidence of 1 in 10,000. There was of course no disagreement with the view that dietary deficiency was one of the important causes.

Further reports on pregnancy anaemia from other parts of India were gradually appearing in the medical journals. Mudaliar and Rao (1932) reported on 32 cases in Madras, 20 of which were treated with marmite with disappointing results. They recommended liver in sufficient amounts for treatment. The authors stated that the diets of these patients were lacking in fresh green vegetables and fats, and hence in vitamins A, C and D. They believed that a constitutional factor, a factor associated with pregnancy and an antianaemic factor were together responsible for pregnancy anaemia. Gupta (1932) studied the records of 203 cases in Calcutta between 1928-1930 and reported that haematological and histopathological findings at autopsy were similar to those in Addisonian anaemia. On the other hand, Mitra (1931) from study of 86 cases in Calcutta expressed the opinion that anaemia was toxaemic in origin. Mitra (1937) later described a further series of 165 cases nearly half of which were macrocytic. Napier and Das Gupta (1937) observed that the incidence of all anaemias in tea garden labour women was 10 per cent and that it tended to increase in pregnancy. The incidence was highest in the age group 15 to 20 years. Chowdhury and Mangalik (1938) find an incidence of 5.6 per cent among the women confined in a hospital at Agra in 1936-1937. They examined only 41 cases of which 27 were of the macrocytic variety. They suggest that toxaemia of pregnancy results from anaemia and that proper anti-anaemic



treatment prevents the appearance of toxaemia or causes its disappearance. Upadhyay (1944) has paid detailed attention to the dietary history of normal non-pregnant and pregnant and anaemic pregnant women in Patna. He found that the largest number of cases was derived from the subjects living on diets of maize, sweet potato with green vegetables occasionally, fewer from those who included any kind of pulse in their diets and there were still smaller number of anaemia cases from the group which had a more varied diet containing rice, pulse, wheat, vegetables, milk, etc. There were no anaemia cases from the group which took meat, fish, or eggs in addition. In 1940, Napier reviewed the existing knowledge on pregnancy anaemias which in India stood high among the causes of maternal mortality. He considers that a slight degree of anaemia in pregnancy is physiological (Bethell, 1936) but in India the condition is often aggravated by defective dietaries and tropical disease. The microcytic hypochromic anaemia in pregnancy is due to the same factors which are operative for similar anaemia seen in the general population. Some cases of macrocytic anaemias in pregnancy which show evidence of haemolysis may be due to malaria. On the other hand, macrocytic hyperchromic anaemia in pregnancy must be due to some as yet unknown dietary deficiency. Later work already referred to in connection with NMA demonstrated that folic acid was equally effective in macrocytic anaemia of pregnancy. Das Gupta, Chatterjea and Mathen (1949) treated 14 cases with folic acid during pregnancy and 17 cases after parturition. Most of the pregnant cases responded well to 20 to 30 mg./day of folic acid in 30 days. The response in the *post partum* cases was, however, not so good.

Recently, crystalline vitamin B<sub>12</sub> has been tried in macrocytic anaemia of pregnancy. Patel and Kocher (1950) reported good haematological response in four such cases with a megaloblastic marrow. They observe, however, that in both the NMA and macrocytic anaemia of pregnancy the dose is about four times that required for treatment of Addisonian anaemia. This finding is contradictory to that of Ungley and Thompson (1950) who observed that much larger doses of crystalline vitamin B<sub>12</sub>, viz., 65 to 80  $\mu$ g., by parenteral route, were ineffective in six cases of megaloblastic anaemias of pregnancy and puerperium. All these later responded to folic acid, some to even as low a dose as 2.5 mg./day.

It should now be clear that all the different types of anaemias occurring in India have primarily a poor dietary background for their origin. While it is easy to understand the occurrence of normochromic normocytic and hypochromic microcytic anaemias as being due to defective diets with multiple deficiencies on which may or may not be superimposed hookworm disease, while it is not difficult to visualize the occurrence of haemolytic anaemias caused by a disease like malaria, it is not so easy to conceive of specific dietary or other conditions responsible for NMA and macrocytic anaemia of pregnancy which presumably are due to a deficiency of folic acid or vitamin B<sub>12</sub> and possibly also of other related haemopoietic factors. The lack of animal protein can obviously not be the main factor unless it be assumed that it acts by influencing the microflora of small intestines which have the capacity to synthesize folic acid and other haemopoietic factors. Ungley and Thompson (loc. cit) point out that in their cases the dietary histories did not suggest that the intake of

folic acid by the patients was lower than that of other women of the same economic groups. The same, however, cannot be said of the Indian cases. These are derived usually from the class of people whose diets have been repeatedly shown to be inadequate. Under these conditions, other vitamin deficiencies ought to be encountered. Probably they are associated with anaemias of the macrocytic type; unfortunately this aspect has not been referred to by most of the Indian workers on anaemias. On the other hand, such association may not have been so marked as to invite special comment. It is the common experience, however, that even though multiple deficiencies are often present, it is the signs and symptoms of deficiency attributable to one nutrient which somehow predominate. The reasons for this selective manifestation would certainly merit further study.

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## CHAPTER XVIII

### DEFICIENCY DISEASES

IN THE Chapter on Nutrition Surveys a brief reference has been made to the recording of certain signs and symptoms in connection with the attempts made to evaluate nutriture. Apart from such surveys there also exists much published work in India which deals with nutritional diseases. Its study yields a great deal of valuable information on the occurrence of nutritional diseases conditioned by dietary and other factors. Unfortunately such diseases are only too common in India and the wealth of material for clinical study is impressive in quantity and variety. It is, therefore, unfortunate that till recently this abundant material has not been utilised for studies on aetiology, pathogenesis, diagnosis and treatment of nutritional diseases. This has left a wide gap in our knowledge which it is hoped the workers will fill before long.

It is realised that the treatment of this subject in the following pages is far from satisfactory. The arrangement of these diseases here follows neither the etiologic nor the systematic classification. In view of the multiple nature of the deficiencies encountered the variation in their manifestation and the uncertainty as to the etiology of many of them, it was considered desirable to discuss the subject matter in three ways, (a) disorders affecting mainly one system (b) disease entities such as beri-beri, scurvy, rickets etc., and (c) syndromes in which more than one system is involved. The author does not seek to justify this method of presentation; he is merely explaining

the method adopted in dealing with this complicated but intensely interesting subject.

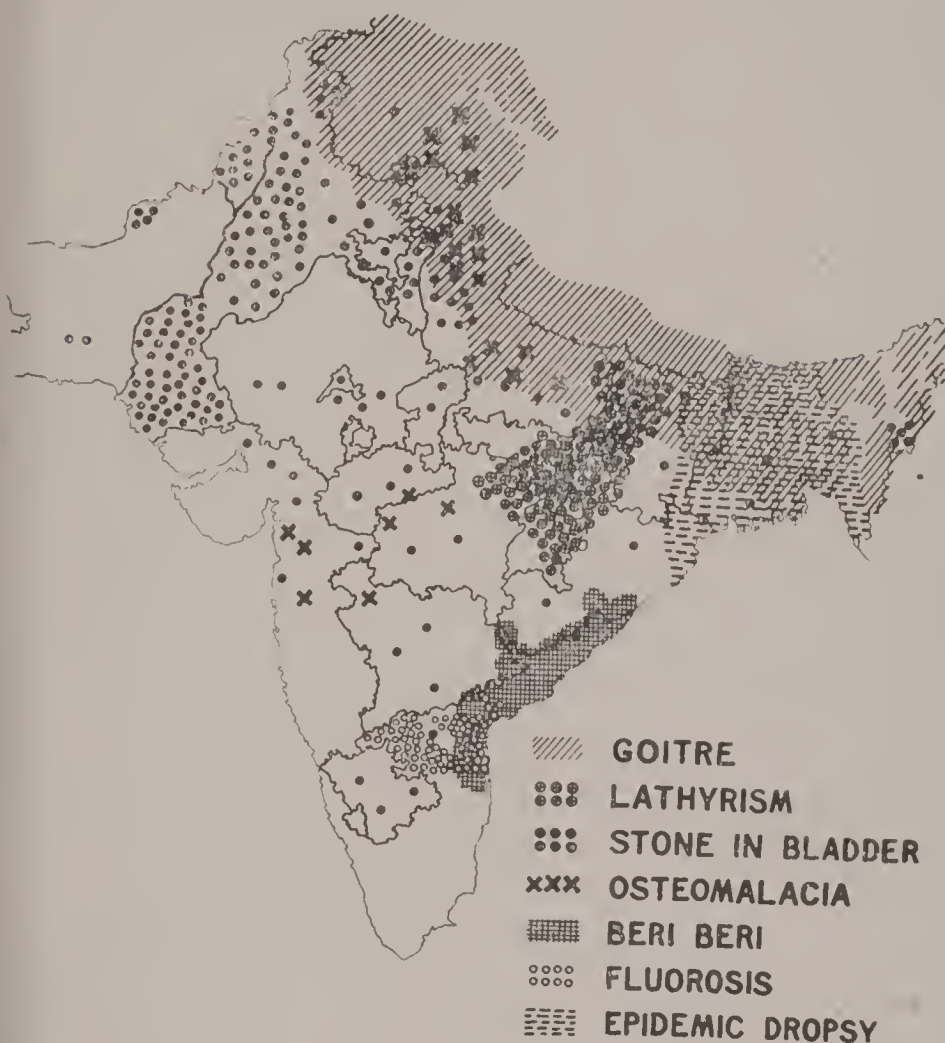


Figure XV—Incidence of diseases associated with faulty diets.

### EYE

**Xerosis:** McCarrison (1920) had stated that xerophthalmia frequently occurs in India among people living on a diet of rice and vegetable oil and that it was curable by cod liver oil. Xerosis of the scleral conjunctiva of varying degrees can be seen in poorer sections of the Indian population. It ranges

from mere dryness and smokiness of the conjunctiva to well marked xerosis and typical Bitot's spots. Aykroyd and Krishnan (1936) have examined 436 children in Bellary district and found evidence of xerosis in 116, nearly 30 per cent of whom showed Bitot's spots. The spots first described by Bitot in 1863 are formed by the heaping up of the degenerated conjunctival epithelium most commonly lateral to the cornea in the equatorial region of the eye.

The spot is either white or cream coloured, triangular in shape with the base pointing towards the cornea. In certain cases the spots may be oval or round. Although in a majority of cases they are situated lateral to the cornea, they may also be found on the nasal side in a few cases. Bitot's spots have been correctly described by Bicknell and Prescott (1947). The description given by Nicholls and Nimal-suriya (1939) of Bitot's spots in Ceylonese children corresponds with the above. Kruse (1941) claims to describe the evolution of what he calls Bitot's spots. Neither the pictures reproduced in his article nor the description of the spots at all point to the lesion being Bitot's spots. According to him the spots are most common on the nasal side of the cornea, are of all sizes and shapes and vary in colour from white to orange. Jolliffe, Tisdall and Cannon (1950) describe the thickened heaped up conjunctival lesion occurring more commonly on the nasal side. Although they do not mention Bitot's spots by name the description is likely to carry the impression that Bitot's spots are being referred to. All that can be said here is that neither their description nor that of Kruse would seem to refer to Bitot's spots. Experience in India almost entirely confirms the original description of Bitot later greatly amplified by Nicholls and





Figure XVI—A Bitot's spot.  
(*Nutrition Museum*)



Figure XVII—Circumcorneal pigmentation—Note the curvilinear pigmentation occurring as concentric ring around the cornea. The ring is drawn away from the cornea in the equatorial region.  
(*By courtesy of the Indian Journal of Medical Research*).



Nimalsuriya, with regard to their location, colour and general appearance (Fig. XVI).

Thickening and folding of scleral conjunctiva and lack of lustre are not uncommon among adults (Ramalingaswami and Patwardhan, 1949). By far the most serious condition arising out of vitamin A deficiency is keratomalacia among children. In fact, Wright (1931) considered it the most important cause of preventable blindness in India. Kirwan, Sen and Biswas (1941) analysed the records of 14,698 patients seen at the Eye Infirmary at Calcutta and observed that 419 or 2.85 per cent suffered from conditions attributable to vitamin A deficiency of varying degrees of severity. In a further communication Kirwan, Sen and Bose (1943) stated that keratomalacia was most common below five years of age, the incidence in Calcutta being 10.5 per 10,000 of population. Heilig (1943) reports having seen in a Mysore hospital 32 cases of keratomalacia in five years in children under five years. Not infrequently cases appear at the Nutrition Clinic at Coonoor as well; 27 cases have been encountered during the last three years. Aykroyd and Wright (1937) have tried orally administered emulsion of carotene in oil in early cases of keratomalacia and have found good response to this treatment. In most cases, however, massive doses of vitamin A in the form of a concentrate have been used for effective treatment of this acute condition.

**Conjunctival Pigmentation:** Patchy pigmentation of bulbar conjunctiva has often been noticed among school children and has been ascribed to vitamin A deficiency. That it is not necessarily so has been demonstrated by Kirwan *et al* who tested the dark adaptation of patients showing patchy pigmentation and found values equal to those obtained with

normals. Dhurandhar and Boman Behram (1940) described a curvilinear pigmentation surrounding the cornea which was seen in rings, semicircles or segments round the cornea. The colour of the pigment varied from gray to light brown or dark brown. They found an incidence of 89.4 per cent in an industrial school where the diets were very poor in protective foodstuffs. On the other hand, in a paying day school the incidence was 26.8 per cent. About 72 per cent of the boys with pigmented conjunctiva had defective vision in one eye or both. In 80 per cent of the latter impaired light sensitivity persisted even after correction of refractive error. Impaired light sensitivity and a large difference in the incidence between two schools led the authors to suggest that the pigmentation was probably nutritional in origin. But Kirwan's findings do not lend support to the above suggestion. Recently a report from Assam by Gilroy (1949) showed that similar pigmentation occurs there on tea plantations in association with xerosis. A survey at Coonoor in 1950 brought to light the incidence of curvilinear pigmentation as high as 80.6 per cent not associated with xerosis and not responding to continued treatment with vitamin A. A nutrition survey in Uttar Pradesh has brought to light this type of pigmentation among school children. It is difficult as yet to say whether the patchy or linear pigmentation (Fig. XVII) observed in Indian children is due to malnutrition or to other as yet unknown causes.

**Night Blindness:** This condition is often met with among the children of poor classes in India. It is possible that it may occur more in certain parts of India than in others, detailed information, however, is not available. At ophthalmic hospitals it must be one of the common complaints. Kirwan *et al* (1943)

investigated 200 cases among which 41 were suffering from disease either of the retina or optic nerve. In 110 cases fundus was found normal, of these 69 showed impaired dark adaptation when tested with the biophotometer. If other causes of night blindness are excluded, the number of patients showing nutritional night blindness was thus not inconsiderable. Kirwan expresses a doubt regarding the deficiency of vitamin A being the aetiological factor in all of these cases. He treated 58 cases of which 20 failed to take the full course of treatment, 37 showed improvement on various vitamin A preparations and only one failed to respond. In view of these results it is difficult to take the opinion of Kirwan seriously. Dhurandhar and Boman Behram (*loc. cit*) also report that 72 per cent of their subjects with defective vision and conjunctival pigmentation suffered from diminished light sensitivity. Heilig (1943) states that in Bangalore and Mysore 12 to 14 per cent of ophthalmic patients diagnosed as vitamin A deficiency cases complained of night blindness.

Rajagopal (1941) tested ten cases of night blindness by the Birch-Hirschfeld type of adaptometer and found that administration of vitamin A in massive doses (216,000 I.U.) produced improvement within six hours and that 48 hours after vitamin A the threshold values returned to normal. Basu and De (1941) found the use of Jeans Zentmire biophotometer unsatisfactory, they thereupon modified the instrument and determined the dark adaptation in 341 school children, among whom 23 per cent were found to suffer from impaired dark adaptation. Among 161 adults a similar result was obtained. They could, however, test therapeutically only seven individuals from the latter group and in these, distinct improvement

in dark adaptation was found on 10,000 I.U. vitamin A given daily for about 40 days. Khan (1945) reports the occurrence of night blindness in Indian troops during the last war. Out of 1,090 Indian soldiers examined by biophotometer in a forward area 300 were found to show impaired dark adaptation. In a few cases where therapeutic tests were made (25 cases only) the condition improved on administration of vitaminised oil. Hassan and Khanna (1947) have compared the vitamin A content of blood plasma in medical students of Lahore and their dark adaptation. They found that values of 50 I.U./100 c.c. and below were associated with impaired dark adaptation, which improved by vitamin A therapy.

Sardana (1946) describes 159 cases of avitaminosis A in a prisoner-of-war hospital in Singapore in 1943. There was no xerosis or keratomalacia, cornea was insensitive in 81 per cent cases. Media were clear and fundi normal. All suffered from impaired dark adaptation and concentric contraction of fields of vision. In advanced stages distant vision was failing even during day time. Treatment was started with red palm oil and bananas, later other foodstuffs containing carotene and other vitamins were used. About 85 per cent cases improved within four weeks.

**Other Conditions:** Verma (1942) describes cases of optic atrophy as seen in children and young adults for which no structural abnormality could account. The only positive finding was the pallor of the temporal half of the optic disc most often in both eyes. The patients were derived from poorest classes, the duration of the complaint was ten months to seven years with insidious onset. In 48 cases which Verma investigated 23 had angular stomatitis, 18 xerosis, and 6 were with tender calves with sluggish knee



jerk. Dried yeast and shark liver oil were given for treatment, the less severely affected cases showed marked improvement whereas in others the response was poor. Verma believes that where optic atrophy has advanced to a certain stage, treatment does not improve matters although associated conditions such as angular stomatitis and xerosis do respond. Aykroyd and Verma (1942) report 13 cases of superficial keratitis with angular stomatitis and fissured tongue as associated signs of riboflavin deficiency. The ocular symptoms consisted of burning of eyes, mistiness of vision, photophobia and lacrimation, circumcorneal injection with opacities in cornea visible on direct or oblique illumination. Vascularisation of the cornea was found in only three patients. The treatment consisted of 2 mg. riboflavin by intramuscular injection on the first day followed by 1 or 2 mg. riboflavin daily by the same route. In 3 to 12 days all subjective eye symptoms disappeared followed by improvement in the physical condition of the eye. Verma (1944) has also successfully treated 20 cases of angular conjunctivitis with riboflavin injections. It could be assumed, therefore, that burning of eyes, dim vision, lacrimation and photophobia, circumcorneal injection with opacities in cornea and optic atrophy etc.,—one or all of these—may have been caused by riboflavin deficiency. It is peculiar, however, that eye conditions attributable to riboflavin deficiency were so common in Madras for Kirwan, Sen and Bose (1944) consider that in Bengal eye disease due to riboflavin deficiency is rare.

Sardana (1946) describes the involvement of the eye in 121 cases of ariboflavinosis among prisoners-of-war in Singapore. The main complaint was haziness of vision in bright light, vision being better in



cloudy weather and at dusk or dawn. Cheilosis and angular stomatitis were present in nearly all cases, and 78 per cent of the cases suffered from seborrhoeic dermatitis 4 to 8 months prior to the onset of eye symptoms. The cornea, media and fundi were clear. In the early stages, upper and temporal visual fields were contracted, in advanced cases all four quadrants were affected. At this stage the patients complained of poor night vision. These patients showed no response to treatment with red palm oil or vitamin A rich supplements.

Sardana also gives an account of 220 cases with ocular involvement in (according to him) thiamine deficiency. The symptoms were dimness of vision at 20 - 30 yards and difficulty in reading. Most of these complained of burning sensation in eyes, pain behind the eyeball, lacrimation, mild photophobia and some degree of blepharospasm. Visual disturbance in day-time and night was equally marked. It is possible that some of these symptoms may have been due to associated riboflavin deficiency. Conjunctiva, cornea, media, and fundi were normal except in 2 per cent who showed optic nerve atrophy along with other symptoms of advanced dry beri-beri. Visual field showed mild peripheral contraction, accommodation was weak and tiring.

As pure vitamins were not available these cases were treated with foodstuffs rich in members of the B-complex. There is no doubt that the ocular conditions described by Sardana differ among themselves. On the basis of (a) associated conditions attributable to deficiencies of vitamin A, riboflavin and thiamine and (b) the results of limited therapeutic trials the major responsibility for their incidence can be assigned to the above vitamin deficiencies. It is probable,

however, that the whole clinical picture may have been modified by coexistent deficiencies of other members of the B-complex even if it is conceded that the ocular symptoms arose predominantly due to individual deficiencies. It is indeed interesting to note the very careful description given by Sardana stressing finer points of difference between the three conditions particularly where the impairment of sensitivity to light, reaction of the pupil and contraction of visual fields are concerned. Unfortunately the resources for treatment were extremely limited in a prisoner-of-war hospital and do to a certain extent detract from the value of these detailed observations.

### SKIN

**Dry and Rough Skin:** Among the poor class children especially in South India, one is struck with the common occurrence of dry and rough skin. The natural gloss is absent, the skin is rough to the feel, elasticity is diminished, and not infrequently there is branny desquamation. Association with xerosis of the eye has been observed by Gopalan (1950) and with phrynoderma by Menon, Tulpule, and Patwardhan (1950). Although dry and rough skin has been included as an item in most nutritional survey schedules in India, the investigators in the past have been silent about its occurrence. The condition is usually attributed to vitamin A deficiency but the observations of Menon, Tulpule and Patwardhan (loc. cit) suggest that dietary deficiency of fat may have something to do with this abnormality. Aykroyd and Rajagopal (1936) have commented upon the absence of dry and rough skin in Malabar where oil inunction is common.

**Phrynoderma (Follicular Hyperkeratosis):** This is a condition manifested as raised horny papules with hyperpigmented summits distributed bilaterally on the lateral and posterior aspects of lower and upper extremities. They are numerous on the thigh, the arm and buttocks. In advanced cases they may also be found on the forearms, legs and trunk (Fig. XVIII).

Radhakrishna Rao (1937) studied the histopathology of the lesion and found that the papules arose from the pilosebaceous follicles. A noninflammatory hyperkeratosis of the lining epithelium causes a keratinous plug to form at the mouth of the follicle and leads to its distension. Atrophy of sebaceous glands was secondary and due to pressure. The absence of perifollicular vascular changes or haemorrhages in the tissue distinguishes this condition from an almost similar one seen in ascorbic acid deficiency.

The condition was described by Nicholls (1933) and given the name phrynoderma or toad skin. He first saw it among indentured East African labourers and later among the Ceylonese children. Later on numerous observers all over the world reported the occurrence of phrynoderma as a deficiency condition. The opinion originally expressed by Nicholls that it was a manifestation of vitamin A deficiency has found popular support although it has been questioned by several workers. Gopalan (1947) has reviewed the evidence for and against the hypothesis of vitamin A deficiency being the causative factor. Earlier, Aykroyd and Rajagopal (1936) had made a suggestion that phrynoderma may be due to deficiency of fat. It was Gopalan, however, who first treated groups of cases with linseed oil, yeast extract—singly or together—and vitamin A concentrate respectively. He found poor response to vitamin A; linseed oil brought about



Figure XVIII—Phrynomeris: The extensor aspects of the extremities are involved. The horny summits of the keratinized follicles may be seen clearly in the magnified picture on the left.  
(*Nutrition Museum*).





distinct improvement but a combination of the oil and the yeast extract gave the best results. Gopalan also observed that in 112 cases of phrynoderma seen by him only four had xerosis as well, whereas 94 had associated symptoms attributable to B<sub>2</sub> complex deficiency. This observation together with the above mentioned therapeutic trials strengthened the belief that fat deficiency complicated by a deficiency of one or more members of vitamin B<sub>2</sub> complex had something to do with phrynoderma. Three years later, Menon, Tulpule and Patwardhan (1950) provided additional proof in support of the aetiological role of fat deficiency. They found that children of comparable age groups living on identical diets but showing phrynoderma had serum fatty acids which were more saturated than the serum fatty acids of children not showing this condition. They treated 48 cases with sesame oil and marmite singly or together and observed good response to the oil alone. In all cases which responded, the unsaturation of serum fatty acids rose to the normal. Hansen *et al* (1933, 1936 and 1937) had demonstrated the relation between the iodine value of serum fatty acids and fat deficiency and Nhavi and Patwardhan (1946) had observed low unsaturation of serum fatty acids in persons living habitually on very low fat intakes. The fact that the diet of the patients studied by Menon *et al* was unchanged throughout the treatment (the patients were the inmates of an orphanage) and that no vitamin A or its precursor was included in the treatment makes it fairly clear that vitamin A is not involved in the aetiology of phrynoderma. The chain of evidence with regard to fat deficiency hypothesis is not yet complete. However, the evidence produced above is strongly in favour of either fat deficiency or

disturbance in fat metabolism being the aetiological factor in phrynoderma.

The incidence of phrynoderma in India as observed in nutrition surveys has been illustrated in figure XIV on page 169. The very low incidence in Bengal, Assam and Delhi is noteworthy. Although extensive surveys have been done in Hyderabad State no mention seems to have been made about phrynoderma. It is too much to expect that the condition is completely absent in children in that region.

**Other Conditions:** Gopalan (1950) has listed a few other conditions of the skin and its appendages seen by him as clinical manifestations of malnutrition. Among them may be mentioned the following two: (a) lichenification of skin over the knuckles and dorsum of hand, elbow and on the ankle joints, (b) diffuse hyperpigmentation resulting in darkening of complexion and of skin over the ends of extremities; this was necessarily associated with pellagrous symptoms.

There are several deficiency diseases in which skin is affected. Since the involvement of the latter forms part of certain well known syndromes, which are going to be described in detail later, the description of skin conditions associated with them will be deferred to the appropriate place.

### ALIMENTARY SYSTEM

**Angular Stomatitis:** Aykroyd and Krishnan (1936) first described the occurrence of angular stomatitis in India among school children in the vicinity of Coonoor. The authors mention that this type of stomatitis was rather common in children in South India and was probably similar to *perleche* of the French writers. Aykroyd and Krishnan reported successful

therapy with dried yeast and skim milk powder. Two years later they treated another batch of boys with yeast autoclaved at  $130^{\circ}\text{C}$  and at pH 9.2 for five hours also with good results, hence they concluded that stomatitis was not due to thiamine or riboflavin deficiency. In the United States, Sebrell and Butler (1938, 1939) described similar lesions in a group of women placed on experimental diets in whom angular stomatitis responded to administration of riboflavin alone. These observations could explain the earlier ones of Aykroyd and Krishnan wherein subjects obtained relief with dried yeast. It is, however, difficult to reconcile their later observation on the effect of autoclaved yeast extract from which riboflavin was believed to have been destroyed. In the same category can be placed the report of Passmore, Sommerville and Swaminathan (1940) on five cases of angular stomatitis with porphyrinuria who improved on treatment with 100 to 400 mg. of nicotinic acid per day. However, observations are on record (Darby, 1950) which indicate that angular stomatitis may occur in nicotinic acid deficiency, in which case the above discordant observations could be satisfactorily explained.

Karunakaran and Nair (1940) have reported on 92 cases of deficiency disease in an asylum in Travancore. Of these, 81 had scrotal eczema, 60 showed angular stomatitis and 58 glossitis which appears from their description to have been of atrophic type. All the three conditions responded to treatment with marmite or butter milk. The authors also tested the efficacy of cod liver oil and found that it caused a certain improvement in scrotal eczema but complete cure was obtained only on treating with marmite or butter milk (which is milk soured and skimmed).

Mitra (1943) treated 32 boys showing angular stomatitis, glossitis and scrotal dermatitis with shark liver oil, skim milk, dried yeast, nicotinic acid and riboflavin given separately to different individuals. Skim milk and riboflavin each caused good improvement, but the best and most rapid results were obtained by combining nicotinic acid and riboflavin. Mitra also observed that on nicotinic acid alone, soreness and burning of mouth decreased but angular stomatitis deteriorated. The above two reports show that riboflavin deficiency results in angular stomatitis but that the picture may be complicated by nicotinic acid deficiency and manifest itself as red raw tongue and accentuated dermatitis of the scrotum. That genitals may be affected in ariboflavinosis is shown by two further publications, one by Vakil (1945) and the other by Gopalan (1946). The former describes one case with vulvitis in a girl of 18 showing angular stomatitis as well, both conditions responding to injections of riboflavin. Gopalan lists leucorrhoea, pruritus vulvae, varying degrees of vulval inflammation and vulvo-vaginitis in women of child bearing age and prepuccial ulceration in the adult males as associated symptoms of ariboflavinosis. In all these cases cures had been obtained by riboflavin alone.

**Glossitis:** The involvement of the tongue is common in deficiencies of riboflavin and nicotinic acid, and in macrocytic anaemias. A detailed discussion of the aetiology of glossitis and associated clinical conditions would be outside the scope of this book. In India too, the occurrence of glossitis with or without other nutritional deficiency manifestations has been reported on several occasions. One report, however, deserves special mention. Fitzgerald (1932) reports an outbreak of glossitis in Assam Jail where



one-third of the total population of 400 was affected. The tongue showed patchy desquamation of varying degrees, papillae were either hypertrophied or atrophic, colour pale and glazed or red and raw; fissuring and indentation were also present. Glossitis was accompanied by burning pain on eating and swallowing, abdominal discomfort with pain, eructation, flatulence and diarrhoea. In three cases there was tingling and numbness and in two symmetrical dermatosis. The outbreak occurred in monsoon months, 70 per cent of the cases recovered without treatment, in others one ounce of dried yeast per day brought on cure in 93 per cent of those treated. From the general description it appears as if the glossitis was due to a multiple deficiency of more than one member of the B<sub>2</sub> complex. That 70 per cent of the untreated cases improved spontaneously suggests that this effect may have been due to some change in the dietary which did not receive as close attention as it should have for at that time the relation between deficiency condition and glossitis had not been fully proved.

Glossitis has also been reported in a few nutrition surveys done in India particularly in Hyderabad State where its incidence among children was found to vary between 0.5 to 1.2 per cent. It should be considered significant that in the same child population the incidence of angular stomatitis was between 1.1 to 6.6 per cent. Whether both these conditions existed in the same children is difficult to decide in the absence of any details.

**Stomach-Gastric Ulcer:** Attempts have been made in India to correlate the deficient dietaries and the incidence of peptic ulcer. McCarrison (1931) fed two groups of rats diets consisting of rice or a mixture of equal parts of rice and tapioca to which were added



fish, chillies and tamarind. The diets were so devised as to be similar to those consumed by human beings in Travancore, where peptic ulcer was believed to be fairly common. Out of 18 rats kept on rice and tapioca diets seven developed ulcers 0.5 to 1.0 cm. in diameter either in the squamous or the mucous regions of the stomach and three showed symptoms of gastritis. In the rice groups only two showed gastric ulcer. McCarrison believed that tapioca diet predisposed to gastric ulcer. Rao (1938) found that in patients of gastric ulcer of  $1\frac{1}{2}$  to 15 years duration the bisulphite binding substances (B.B.S.) of blood showed a substantial increase over the normal values. The pyruvic acid content of blood formed 2.6 to 96 per cent of the total B.B.S. From these results Rao concludes that in peptic ulcer cases a vitamin B<sub>1</sub> deficiency exists. This may well be true, but the B<sub>1</sub> deficiency may be due to continued dietary restriction either voluntary or under medical advice, for such secondary deficiencies are known to occur in several chronic diseases. Rao (1938) determined the blood level of ascorbic acid of proved peptic ulcer patients and found it between 0.40 to 1.92 mg. per cent, i.e. within the normal range; he, therefore, does not believe that vitamin C deficiency has any aetiological role in peptic ulcer. On the other hand, Rao (1937) is of the opinion that the hyperacidity and hypermotility of stomach seen in normals in Vizagapatam may be one of the predisposing factors. His suggestion was based on the results of an investigation on gastric function of 100 healthy normals. In 80, he found higher values for the range of initial acidity and maximum acidity. Pepsin content of the juice and the motility of stomach were also higher as compared to Ryle's series. In a further communication Rao

(1939) concludes that spices in Indian diets do not cause continuous irritation since no abnormal quantities of mucus were found in gastric juice specimens. He argues that probably lower protein and B-complex content of diets together with hyperacidity act as predisposing factors for peptic ulcer. There is no further evidence forthcoming in support of either of these hypotheses. On the other hand, recent evidence (Lancet, 1949) shows that hyperacidity does not damage gastric mucosa and that on biopsy of gastric mucosa through gastroscope, lesions heal as rapidly in subjects with hyperacidity as in normals.

**Intestines :—**Diarrhoea as a symptom of nutritional disorder has been encountered in children as well as adults. Earlier experimental work of McCarrison (1921) had indicated the relationship between poor diets and intestinal function and integrity of its structure. His extensive researches had demonstrated such effects as atrophy of the myenteron, degenerative changes in the plexus of Auerbach, atrophy and inflammation of the mucous membrane, etc., in pigeons, guinea pigs and monkeys. Rao (1942) fed young monkeys on poor South Indian rice diet and produced similar lesions in the intestinal tract. Aykroyd (1942) had pointed out that the recorded death rate from dysentery and diarrhoea in Punjab was usually  $1/4$  to  $1/6$  that prevailing in certain southern provinces of India. He thought that the better dietary of Punjabis was probably responsible for the low incidence of lethal gastrointestinal disorders.

Heilig (1943) has reported upon the successful treatment of nonspecific diarrhoeas with nicotinic acid in Mysore hospitals. From Gujerat, Cook (1944) describes 50 cases of watery diarrhoea in malnourished people showing the associated signs and symptoms

of malnutrition such as weight loss, glossitis, paresthesias, anaemia, oedema of legs and pigmentation of pellagrous distribution. The cases responded to nicotinic acid, which controlled the diarrhoea within 48 hours. Cook believes that diarrhoea in his cases was indeed a manifestation of malnutrition, for poor people in Gujerat live on diets rich in carbohydrate and poor in protective foodstuffs. According to him the condition is fairly common in Gujerat among such people and is the cause of much ill-health. Aykroyd and Gopalan (1945) investigated in detail 54 cases of chronic diarrhoeas in Madras. They eliminated other causes by a careful clinical investigation together with microscopic and cultural examination of stools. The condition described by them was insidious in onset and had started with occasional loose stools. In severe cases 10 to 12 stools were passed per day. There was no abdominal pain or griping and no tenesmus, although general abdominal discomfort was present. Sore tongue was present in 49 cases and dependent oedema in 25, glossitis appearing at about the same time as diarrhoea and oedema about a couple of months later. In 36 cases there was atrophic glossitis with fissured tongue while in 13 the glossitis was of the hypertrophic variety. In 15 there was 'crazy pavement dermatosis' unlike that found in pellagra. In 28 cases there was achlorhydria (action of histamine not tested) and in 15 hypochlorhydria. Response to a daily administration of 50-100 mg. nicotinic acid intramuscularly was rapid, the improvement being apparent after the third injection. In all the 50 cases diarrhoea stopped within 12 days. Oedema disappeared only after the patients were placed on hospital diet subsequent to the control of diarrhoea. Glossitis improved only slightly and no

striking change in dermatosis was noticed. Eleven cases returned to hospital within two months after discharge and were again cured with nicotinic acid. The authors believe that rapid improvement on treatment cannot be ascribed entirely to the restoration of the normal condition of the possibly atrophied mucosa, but that it suggests the correction of some functional disturbance in the nervous control of intestinal peristalsis. Singh (1945) reports on 26 cases of fatty diarrhoeas in Indian troops in a forward area probably as a result of dietary restriction. Fat in the stools varied from 38 to 71 per cent of which split fat was 37.5 per cent. Nicotinic acid orally was found ineffective. The patients were treated with good hospital diet and sulphaguanidine. Autopsy findings in three fatal cases were generalised atrophy of intestinal musculature with fibrosis in subepithelial and submucous tissues.

**Sprue:** There have been several investigations on sprue in India. There was a belief current in the early days that only Europeans in India suffered from sprue and that Indians were immune from attack. Fairley and Mackie (1926) remarked that although the former may be the case the latter was not necessarily correct. These authors were of the opinion that sprue might be a virus disease, a conclusion we now know not to be correct. They investigated gastric and pancreatic function and found both to be normal. Blood picture revealed macrocytic anaemia and the bone marrow a megaloblastic reaction. Sokhey and Malandkar (1928) concluded on the basis of stool analyses that pancreatic function in sprue was not affected. Mackie and Fairley (1929) studied the pathology of intestinal tract in eight cases dying of sprue and were of the opinion that sprue



was a disease of the intestinal tract which, if progressive, resulted in the degeneration and destruction of the absorbing and secretory function leading to slow starvation. Anaemia and other manifestations of the disease were, according to them, due to the absorption of toxins from the damaged intestinal mucosa.

Ramanujayya (1930) described five cases of sprue from Madras. Malcomson and Murthy (1931) had also reported the occurrence of sprue among Indians in Madras. Hance (1930) treated 26 cases of chronic diarrhoeas in Kathiawar of which 20 were of typical sprue. Therapy was so mixed consisting of autovaccine, emetine and kurchi extract, dilute hydrochloric acid and liver diet of Minot and Murphy that it is difficult to assess its value. The author was no doubt motivated by (a) Hurst's hypothesis of intestinal ulceration by haemolytic streptococci and streptococcal toxins and (b) finding of *Entamoeba histolytica* cysts in majority of cases, since he considered that both these may be predisposing factors in sprue. Chaudhuri and Rai Chaudhuri (1944) described 22 cases of sprue—like diarrhoea from Calcutta. The patients suffered from indigestion, flatulence, anorexia, asthenia, loose irregular motions at any time of the day, glossitis, macrocytic anaemia and emaciation. In all of them there was a history of dysentery or colitis treated unsatisfactorily and causing restriction of diets for prolonged periods. In 15 cases faecal fat was 25 to 33 per cent on a standard diet containing 100 gm. fat. Glucose tolerance curve was flat. Patients were kept on liquid diet followed by the solid hospital diet as the patients responded to treatment. Crude liver extract injections were given. The authors distinguish the condition from sprue by



calling it para-sprue. Even if this distinction is accepted the syndrome appears to be closely related to sprue in most respects. Karamchandani and Hyder (1946) treated 242 cases of sprue in Indian troops at a hospital during World War II. Of these, 31 had amoebic infection as well. The treatment was with raw liver juice from eight ounces of liver or with liver extract parenterally when the latter was available. The cure was complete in 129 patients, 22 showed no improvement and the remainder improved to varying degrees. Detailed studies on sprue in India in more recent times are, however, rare. Black and Fourman (1945) examined over 150 stools of military patients admitted to hospital clinically diagnosed as sprue. The stools were examined three days after the patients were kept on a diet containing 70 gm. fat per day. In 102 stools, fat content was 25 to 40 per cent. Fat globules were seen in only 3, soap plaques in 7 and fatty acid crystals in 55. The authors are of the opinion that mere microscopic examination is not enough to distinguish between pancreatic steatorrhoea and sprue stools, chemical examination being necessary.

It is somewhat surprising that after the appearance of folic acid on the scene there have been no further reports on sprue with the exception of one by Ramalingaswami and Menon (1949) who describe the treatment of one case with folic acid. They found that although there was clinical improvement accompanied by the amelioration of macrocytic anaemia, the haemopoietic response did not proceed beyond a certain stage. Further response was only obtained when proteolysed liver was given. They thus confirmed the observations of Davidson, Girdwood and Innes (1947) and of Morrison and St. Johnston (1947).

A good account of sprue has been published by the Medical Directorate of the Indian Army (1948). A sprue research team was formed in 1945 to investigate the large number of sprue cases occurring among the British personnel engaged in fighting on the Burma Front. Although over a thousand cases of sprue were seen, detailed investigations were limited to 80 only owing to various circumstances beyond the control of the investigators. In these eighty cases the studies covered careful clinical examination, x-rays with barium meal, biochemical investigations involving analyses of faecal fat and serum lipoids and experiments on fat absorption, etc., and evaluation of treatment with diets, yeast extracts and liver preparations.

The disease occurred among the British personnel from within six months to over three years of service in India. The largest number of cases gave a history of onset between 1 to 2 years of service. The cases presented a varied picture from classical sprue syndrome to less typical features such as steatorrhoea without glossitis or *vice versa*. The most common symptoms were disturbances in appetite, diarrhoea, weakness and loss of weight accompanied to a slightly lesser extent by flatulence, glossitis, heart burn and abdominal distension. The condition was marked by frequent remissions and relapses. In the early stages remission could be brought about by hospitalisation and good diet. This was followed by relapse when the individual returned to duty. The history of dysenteric disorders was no more common in sprue cases than in 340 non-sprue control cases. The blood picture gave evidence of macrocytic anaemia in 25 per cent, macrocytosis alone with normal haemoglobin level and R.B.C. count in 30 per cent and hypochromic anaemia in 9 per cent. The x-ray findings

revealed obliteration of mucosal pattern in the jejunum and bolus formation with aggregation of barium in large lumps in the coils of small intestine. Gross variations in the rate of passage through the intestinal tract were recorded. A reference to faecal fat studies by Black and Fourman (1945) has already been made. The rate and the extent of fat absorption as indicated by chylomicron technique were much reduced. Defects in carbohydrate and protein absorption were also noticed. A majority of patients were treated with high protein hospital diets in which was incorporated cooked liver with or without a supplement of yeast extract. Parenteral liver therapy was needed only in severe cases and in those not responding to dietary treatment. The response to dietary treatment alone was good, but as the cases were invalided to England and not followed up it is difficult to say (as the report admits) if remission could be considered as a cure.

In conclusion it is mentioned that sprue might exist with steatorrhoea and loss of weight as the only presenting symptoms. Those which appear later, such as diarrhoea, anaemia, glossitis, etc., may result from secondary deficiencies. The factor which is responsible for defect in fat absorption and steatorrhoea is unknown though it could be folic acid. The report is an excellent example of the results which could be achieved by team work in the study of a disease syndrome and would well repay careful study.

Ramalingaswami, Venkatachalam and Menon (1948) have described a condition among children with diarrhoea, oedema and dermatosis with or without associated symptoms of B-complex deficiency. They believed that the syndrome was identical with Kwashiorkor or Malignant Malnutrition described by

Williams, Trowell, Gillman and others from different parts of the African Continent. This syndrome has attracted considerable notice in India recently and will be treated elsewhere in greater detail.

Another interesting report on nutritional diarrhoeas among children is that of Ramalingaswami (1948) which describes diarrhoea as a manifestation of vitamin A deficiency. The patients were children between  $2\frac{1}{2}$  to 9 years of age, derived from labour classes in the Nilgiris, particularly from the tea plantations. Twenty cases were investigated and treated. Diarrhoea was of 1-2 months' duration and watery in type accompanied by Bitot's spots in 13 and keratomalacia in six patients. The author also mentions the presence of generalized dry skin in 18, phrynoderma in 8, and dry brittle and lustreless hair in 18 patients; seven had pitting oedema. A careful microscopic examination of the stools was done to eliminate any organic or specific cause for diarrhoea. Fifteen cases were treated with 1 c.c. vitamin A concentrate (Prepalin) equivalent to 72,000 I.U. of vitamin A. Diarrhoea was controlled within 48 hours. The treatment was continued for seven to ten days after which the children were put on shark liver oil. Keratomalacia and Bitot's spots responded very well although follicular hyperkeratosis did not improve. Three cases were treated with kaolin and bismuth carbonate, diarrhoea was reduced but still continued even after three days. In two cases treated with sulphaguanidine diarrhoea was not affected at all. When all these five were placed on vitamin A concentrate the diarrhoea was controlled within 48 hours as in other patients of the series. The role of faulty diets in causing degenerative changes in the intestinal mucosa has been discussed above. It is

probable that in the condition described by Ramalingaswami, there was present a multiple deficiency, but it is not to be doubted that the predominant deficiency was that of vitamin A. The presence of chronic diarrhoea must have superimposed a secondary deficiency on the already existing one and brought about changes attributable to severe vitamin A deficiency such as keratomalacia and Bitot's spots.

### NERVOUS SYSTEM

**Sensory System :** The involvement of the sensory system resulting in loss of sensation, hyperaesthesia and paresthesia is not infrequently seen in diseases due to deficiency of vitamins of the B-complex. In the section on diarrhoeas reference has been made to the fact that disturbances in sensory function were often complained of by a fair proportion of patients. Disturbances resulting from lesions of a major sensory nerve such as the optic nerve have been described under the section dealing with the eye. Under the present section will be discussed only the "burning feet syndrome."

Peraita (1942) in Spain had described a paresthetic causalgic syndrome among the victims of famine. Spillane and Scott (1945), Harrison (1946) and Simpson (1946) had all described the syndrome in the Eastern theatre of World War II. Simpson observed that scrotal and oral lesions of ariboflavinosis preceded the appearance of burning feet. The pain was localised to the metatarsophalangeal region on the plantar surface and was bilateral. It was worse at night causing insomnia. In some cases the pain spread over to the dorsum of the foot also. He found thiamine and vitamin A ineffective but yeast or *Phaseolus radiatus* brought about a cure in 4 to 6 weeks,



and other symptoms improved too. Stannus (1944) also seemed to be of the opinion that burning feet was a manifestation of ariboflavinosis. Gopalan (1946) whose paper appeared about six months earlier than Simpson's has carefully distinguished burning feet from peripheral neuritis of B<sub>1</sub> deficiency. Gopalan also obtained cure in 13 cases with a marmite-like yeast preparation. He, however, went a step further and tried the therapeutic effect of a single member of the B-complex at a time. Thiamine, riboflavin, and nicotinic acid were without effect. Taking his cue probably from the observations of Philips and Engel (1939) and of Wintrobe *et al* (1940, 1942) that pantothenic acid deficiency may cause lesions of spinal cord, he tried this vitamin in his patients. Ten cases were treated with calcium pantothenate by daily intramuscular injections of 20 to 40 mg. per day. Complete cure was obtained in three weeks in all these cases. The burning sensation disappeared first followed by the disappearance of hyperidrosis. The feeling of "pins and needles" was the last to disappear. In discussing the mechanism of the syndrome, Gopalan seems to be undecided between it being due to (a) abnormal and excessive stimulation of the peripheral sensory nerve ending by certain accumulated products of deranged metabolism and (b) vasomotor disturbances.

The diagnosis of burning feet syndrome must be difficult mainly because reliance has to be placed on subjective symptoms described by the patient. There is no method of objectively diagnosing the condition. Lipmann and Kaplan (1946 a, 1946 b) had shown that coenzyme A concerned with acetylation *in vitro* contains large amounts of pantothenic acid. Riggall and Hegsted (1948) found a decrease in acetylation

of para-aminobenzoic acid by pantothenic acid deficient rats. When pantothenate was administered there was a restoration of the rats' capacity to acetylate para-aminobenzoic acid. Sarma, Menon and Venkatachalam (1949) observed that in patients of burning feet syndrome there was a similar lowering in acetylation of para-aminobenzoic acid which improved on treatment with pantothenate but not with riboflavin. These authors suggest that a test could be developed along these lines to aid in the diagnosis of the syndrome.

The therapeutic effect of pantothenate was most remarkable and leads one to the assumption that burning feet syndrome must be a result of pantothenic acid deficiency. That there was an associated deficiency of other members of the B<sub>2</sub> complex, particularly that of riboflavin, can be seen by the other signs and symptoms which accompanied burning feet. It is, however, difficult to explain how on a diet generally poor in B vitamins, as the poor South Indian diets are, deficiency of a minor member of the family becomes so predominant, as to be the chief cause of complaint. We are aware of the fact that this is not an isolated instance, if macrocytic anaemias are considered to be due to deficiency of folic acid and/or vitamin B<sub>12</sub> they would be other examples of a minor vitamin creating a major deficiency. Probably our conception of a minor vitamin requires reorientation.

**Motor System-Spastic Paraplegia:** Gopalan (1950) has reported 61 cases of spastic paraplegia from Madras for which no definite cause could be found. The patients were invariably from the poor classes with a very unsatisfactory dietary history. They did not suffer from any systemic nervous disease nor was

there any evidence of local lesions of spinal cord or its meninges. Kahn and Wasserman tests on blood and cerebrospinal fluid were negative; the cell count, protein, chloride and sugar of cerebrospinal fluid were within normal limits. The subjects were adults, the onset in most insidious but in some sudden. The patients complained of stiffness and weakness of the lower limbs and in advanced cases were unable to use them. A few cases complained of burning feet. There were no other signs of sensory disturbance nor any associated signs and symptoms of malnutrition. Cranial nerves were normal, upper extremities were normal. The loss of power and spasticity was confined to lower extremities only. The abdominal reflexes were normal and in some cases brisk. The knee and ankle jerks were exaggerated with ankle clonus in several and muscles were spastic. Plantar response was extensor. No cause could be found for this syndrome. The only connection it may have with nutrition or diet is the observation that a similar syndrome called Lathyrism (to be described later) is associated with the consumption of Lathyrus pulse. Further, certain observers have noticed spastic paraplegia in prisoners of war held by the Japanese (Spillane 1947). Spillane thinks that the condition originates in malnutrition although the mode of involvement of the pyramidal tract is unknown.

There is one difference between the cases reported by Gopalan and those referred to by Spillane which must be mentioned here. Apart from a few of Gopalan's cases complaining of burning feet none in this series seems to have suffered from other disturbances in the sensory nervous system whereas in prisoners-of-war in the Far East spastic paraplegia cases had a varied and prolonged history of sensory

disturbances and also of malnutrition. Thus there is a greater probability of malnutrition being the contributory factor in the latter than in Gopalan's cases. However, in absence of any conclusive evidence it would be advisable to let the matter rest till more information becomes available.

### BERI-BERI

The rice eating population of India amounts to more than half of the total population and yet beri-beri is endemic in certain restricted areas only. Of these the most important are the districts of Ganjam, Vizagapatam, Godavari, Krishna, Guntur and Nellore—all in the old Madras Presidency. Apart from these, a few circumscribed areas in Bengal and Assam are also endemic for beri-beri. Isolated and sporadic outbreaks of beri-beri have not infrequently been reported from other parts of India as well.

The occurrence of beri-beri in Madras was first described by Malcomson in 1835, i.e., long before the advent of rice mills. According to him beri-beri prevailed extensively in the north-east coast division of Madras Presidency and emigrants from the endemic areas were also prone to the attack of this malady in whatever part of India they migrated to. In a detailed survey of the beri-beri problem in relation to rice, McCarrison and Norris (1924) not only confirm the endemicity of beri-beri in the above mentioned districts of Madras Presidency but they throw additional light on its relationship to rice and rice diets. According to them beri-beri outbreaks outside the endemic areas were few and confined to troops, jails and emigrants from the endemic areas. Beri-beri prevailed in the Madras Presidency for the most part in localities where raw rice, whether home-pounded or milled, was



in general use. Where parboiled rice was in use beri-beri was practically unknown. In endemic areas, consumers of parboiled rice did also suffer from beri-beri but the incidence in these people was far less than in those habitually consuming raw rice. Besides, people living on millets such as cholam or ragi in addition to rice may be susceptible to beri-beri. McCarrison and Norris also draw attention to the fact that although in Madras Presidency there was a rough parallelism between beri-beri incidence and distribution of modern rice mills, there was none in the rice eating regions of Bombay Presidency. The authors were drawn to the conclusion that apart from rice the other constituents of diet played an equally important part in preventing the onset of beri-beri. In this they may be correct. One observation of McCarrison and Norris throws considerable light on the somewhat puzzling finding that beri-beri was common even in persons consuming home-pounded rice. They refer to the practice of keeping cooked rice covered with water overnight intended for consumption the next day. Some people drank the steep water, but poorer people consuming inferior grades of rice preferred to throw away the smelly liquid. Washing of rice in repeated changes of water prior to cooking is a universal practice among rice eaters. Recent researches have shown that washing removes a substantial part of thiamine contained in rice. Thus it is possible that the practice of thorough washing coupled with allowing the cooked rice to steep overnight may remove thiamine to a considerable extent. In the poor man's dietary there was little chance of its being replaced by other articles containing sufficient amounts of thiamine. Hence it is not surprising that among the population where



the diets were particularly poor, beri-beri should be endemic as most of the thiamine in rice had been removed by washing, cooking in excess water and steeping.

That beri-beri still continues to be a public health problem in the endemic areas of Madras Province is borne out by several reports published during the last thirty years. Coyne (1927) describes his experience of the disease in Krishna District. In the district hospital at Guntur, Mahadevan and Raman (1930) encountered over 100 cases per year between 1926-1929. According to these authors beri-beri cases were more numerous in rice eaters, although millet eating population was not exempt from the disease. Within the district, more cases appeared from areas where modern rice mills were concentrated than from other regions, an observation which seems to confirm that of McCarrison and Norris referred to earlier. Aykroyd *et al* (1940) drew attention to the higher incidence of beri-beri in the Northern Circars (the old north-east coast division of Madras Presidency) than in the rest of the province. The figures are given in Table XXII. Data collected for five districts of the Andhradesa in Madras State, viz., Nellore, Krishna, East and West Godavary and Vizagapatam over a period of 1945-49 show that endemicity of beri-beri in these areas is as widespread as ever. Table XXII has been modified to include the above information. The endemic areas are shown in Figure XIX.

It is true that mortality from beri-beri is nothing like it is in some South-East Asian countries, but the high incidence by itself should be considered disturbing enough. Aykroyd and Krishnan (1941) also compared the infantile mortality rates in three towns

from beri-beri area with three other towns in the rest of the province. The average rates for the period 1935 - 39 were higher in the endemic area. Of the total mortality from birth to 12 month period, the

TABLE XXII

### INCIDENCE OF BERI-BERI IN NORTHERN CIRCARS AND THE REST OF MADRAS STATE

Year	Northern Circars (Population 13.9 millions)		Remainder of Madras State (Population 32.9 millions)	
	Cases	Deaths	Cases	Deaths
1932	16,091	93	665	3
1933	20,513	55	822	2
1934	21,849	42	711	5
1935	34,639	89	382	8
1936	20,814	94	452	7
1937	27,488	65	748	5
1938	32,717	64	649	4

Year	Cases of Beri-beri in Madras State	
	Five Districts of Andhradesa	Twelve other Districts
1945	15,664	754
1946	16,782	683
1947	27,210	854
1948	28,281	907
1949	29,061	444

highest was between 2 to 5 months, the peak being reached at the fourth month. This is approximately the period when most cases of infantile beri-beri occur. According to Aykroyd and Krishnan the high mortality between 2 to 5 months is largely made up of deaths due to beri-beri. Very often the mothers



Figure XIX—Beri-beri in Madras State: Shaded area depicts the endemic zone.

(By Courtesy of Indian Journal of Medical Research.)

of these children suffered from incipient beri-beri showing such symptoms as slight paresis, difficulty in walking, numbness and tingling, etc. Deaths at 3-4 months of more than one child in the same family were also revealed on further enquiry. Reddi (1942) has supplemented some of these observations in a later publication.

Krishnan, Ramachandran and Sadhu (1945) treated with thiamine 1,145 infantile beri-beri cases in three years at an Infant Welfare Centre in Coconada. Of these 980 were completely cured; relapses were not uncommon but the infants responded to the same treatment. Pregnant women were given a daily dose of 1 mg. thiamine hydrochloride, this did not altogether prevent the appearance of thiamine deficiency in infants, but did help in reducing the severity of the condition.

It may be worthwhile considering here how it is that beri-beri has still continued as a public health problem in India. The disease has been endemic in one area for well over a hundred years, and has been as fixed as the people themselves and their dietary habits.

The science of nutrition has indisputably shown the relationship between thiamine deficiency and beri-beri. In view of this one should be entitled to ask whether it would not have been possible to eradicate the disease. One will have to go deeper into the cause for a satisfactory answer. The earlier discussion will have shown that endemic beri-beri is due to dependence on rice, faulty cooking practices and the defective dietary habits of the people. Firstly, it cannot be denied that a majority of beri-beri cases occur among the poor classes, although economically favoured groups are not necessarily exempt. Apart

from the fact that faulty cooking practices remove a major proportion of thiamine in rice, the amount available in rice is considerably reduced by milling. Secondly, the poverty of the people precludes the use of other foodstuffs rich in thiamine. People in the endemic areas prefer raw to parboiled rice, the latter variety being consumed, if at all, for only a couple of months during the whole year (Aykroyd and Krishnan, 1941). Besides, parboiled rice is unacceptable on account of the offensive odour and strong colour which such rice almost always has. If the process of parboiling is improved and standardized it may be possible to replace raw milled rice with parboiled rice. The F.A.O. Nutrition Committee of the South-East Asian Countries (1948, 1950) has repeatedly made recommendations to this effect. The American 'conversion' process which is a modern scientifically controlled version of parboiling is unsuitable to the agricultural economy of India and other countries in South-East Asia. For somewhat similar reasons 'enrichment' of rice in India is also difficult to practise. The beri-beri problem is capable of solution not by the above two methods or by affording relief by distribution and administration of vitamin B<sub>1</sub> but by making available at cheaper prices the common Indian pulses which people are accustomed to eat but which the poor cannot afford to buy on account of high prices and their own poverty. Another possible way of combating the disease is to prepare acceptable parboiled rice and through education and propaganda gradually replace the raw milled rice with the parboiled variety. Any attempts designed to improve the economic condition of the people will no doubt help a great deal in the satisfactory solution of the problem.



## PELLAGRA

Pellagra occurs sporadically throughout India, although it is true that more cases have been reported from that part of Madras Province where beri-beri has been known to be endemic. Even there, pellagra does not affect large number of people at one time, but cases do seem to occur continually in extremely small numbers. Thus Raman (1933) saw only four cases in Guntur during four years preceding 1933. Dinkar Rau and Raman (1936) saw between 1933-36 one or two cases per year. In 1940, Raman (1940, 1948) reported on 25 cases and in 1948 on additional 65 cases of pellagra.

Lowe (1931) describes 40 cases seen in the course of six years in the patients of a leprosarium in Hyderabad State. The disease made its appearance in December and was on the increase till the following March. By the month of May the patients either were cured or dead. Patients showed general malaise, anorexia, typical skin lesions, stomatitis and glossitis. Diarrhoea and mental symptoms were comparatively infrequent. Those with mental disorders died. Others seemed to get better without treatment. The latter, when given, consisted of a good diet containing milk, meat, vegetables and fruit supplemented with yeast. The usual diets of the inmates contained no maize although qualitatively they were not quite good. Lowe (1933) later described 30 more cases occurring between December 1931 to February 1932 of which 3 were in nonlepers. In these cases too the diarrhoea and mental disorders were absent. Seven of the patients had similar attacks in the previous year. Treatment consisted of good diet supplemented with brewer's yeast.

From Mysore, Heilig (1943) and Puttaiya (1944) give an account of vitamin deficiencies among hospital patients. The hospital records contain several instances of patients who from the description regarding diarrhoeas, dermatosis, glossitis and in a few dementia, appear to have been suffering from pellagra.

Reports from Bombay Province include those by Mody (1935), Dhayagude and Khadilkar (1939), Caruthurs (1941), Patel and Shah (1942) and Patel and Motashaw (1942), altogether a total of 41 cases over a space of seven years. Cases from Bengal have been reported by Sen Gupta *et al* (1939), Goodall (1940), Napier and Chaudhuri (1943) and Chaudhuri and Chakravarti (1947).

Verma (1943) describes 3 cases from Bihar of suspected pellagra out of which two appear to be cases of genuine pellagra. Nicotinic acid alone or with liver extract gave good response. From Uttar Pradesh (United Provinces) there is a report of an isolated case by Swarup (1930). From Punjab also there are cases described by Gupta (1935), Bajaj (1939) and Kochar (1943). It is only in the cases reported from Kangra Valley by Bajaj that one meets with maize as an article of diet in the pellagra sufferers. Although the author describes only six cases treated with marmite, yeast tablets and liver extract together with changes in diet, he refers to the fact that several such cases occur in the locality. It is felt that a close examination by the public health department of the province would have repaid the trouble.

Aykroyd and Swaminathan (1939) estimated nicotinic acid content of maize from U.S.A., Roumania and India and found values 0.7 to 1.6 mg. per 100 gm. Raw home-pounded Indian rice was found to

contain 2.4 mg. per 100 gm., whereas milled rice contained only 1.6 mg./100 gm. According to these authors the total nicotinic acid content of the poor rice diet did not exceed 12 mg. and was not materially different from the nicotinic acid content of diets based on maize. They could not understand, therefore, the association between maize and pellagra. Later work by Elvehjem and his associates, Woolley, Kodicek and others, has shown the role of maize in inhibiting the utilization of nicotinic acid and it is possible that in human beings subsisting on maize diets with marginal intake of nicotinic acid a deficiency of the nutrient is created resulting in pellagra.

There are a few other biochemical investigations bearing on the subject of pellagra that may be mentioned here. Swaminathan (1939) found in 10 normal subjects a daily urinary excretion of 2.06 to 8.88 mg. total nicotinic acid. Naganna, Giri and Venkatesan (1941) found 4.8 to 6.1 mg. total nicotinic acid per 24 hours in an unspecified number of normals and in the two pellagrins tested it was absent from the urine of one and only 1.8 mg. in that of the other. Kochar (1943) found in six normals a range of 3 to 10.8 mg. On the other hand in 6 pellagrins nicotinic acid in urine per 24 hours varied from traces to 0.69 mg. In normal subjects a test dose of 200 to 400 mg. per day caused a rapid rise in excretion whereas in pellagrins the excretion remained low for days. De and Banerjee (1948) have produced evidence to show that human requirements of nicotinic acid are in the neighbourhood of 8 mg. per day. If one takes into account the lowest intakes of about 12 mg. per day recorded by Aykroyd and Swaminathan (*loc. cit*) in the South Indian diets, there does not appear to be any likelihood of a primary nicotinic acid deficiency among the

Indians. However, nicotinic acid deficiency does appear more often than one realises, if the reports on chronic nonspecific diarrhoeas responding to nicotinic acid are indicative of the situation. It is true, however, that classical pellagra is not one of the commoner nutritional diseases in India.

### SCURVY

In India, scurvy seems to be rare for there are only a few published reports of isolated cases from different parts of the country (Srivastava, 1930, Chakravarti, 1935, Lewis and Dutt, 1942). It is possible that cases treated in hospitals may not have been published. That the disease does occur in times of famine is illustrated by a report from Hissar in Punjab (Khan, 1943). This district in south-east Punjab is periodically subject to famines. The recorded history goes back to 1783, after that famines occurred in 1860, 1869, 1896 and 1899. In none of these has scurvy been recorded as one among the causes of death. In 1938 another famine started, and although the conditions were somewhat better by 1940, semi-famine conditions prevailed for a few years. In a diet survey in 1939 it was found that green vegetables of any kind were completely absent from the diets of the inhabitants. Between December 1939 and March 1940, Khan saw 500 cases of scurvy, some of which were treated either with pure ascorbic acid or with the dehydrated pulp of *Phyllanthus embelica*. In 1940, the inhabitants were given germinated grains to eat after which the incidence of scurvy decreased. After six months the supplement was discontinued when scurvy appeared again and was only controlled after the reintroduction of germinated grain in the diet.

Cook (1945) has recorded his experience in Gujarat with scorbutic epiphysitis in 30 children 11 months to 10 years in age. Scorbutic signs in the gums were seen only in seven patients, and in a few cases subperiosteal haemorrhages were present. Skiagram revealed the presence of scorbutic changes in the epiphyses. According to Cook the condition usually followed debilitating diseases and was often wrongly diagnosed as paralysis; it responded to treatment with ascorbic acid in large doses given orally or parenterally. DeSa (1948) draws attention to the surgical aspects of scurvy. Describing his experience with 20 cases in a children's hospital in Bombay he mentions the fact that nearly half the cases presented themselves in out-patients department as surgical problems. Only routine x-ray of the cases helped to reveal the underlying condition as being scorbutic in origin.

Thus it will be clear that in India scurvy is not one of the common manifestations of malnutrition. This is so in spite of the fact that the usual intake of vitamin C in poor Indian diets is by no means high. As the last two reports indicate, however, scorbutic changes may not be unlikely in the child population and can only be diagnosed by the watchful. On the other hand, it is only natural to expect scurvy to come up as a problem among the population stricken by famine, as the example of Hissar quoted above clearly shows.

#### RICKETS AND OSTEOMALACIA

There seems to be a belief generally current that rickets occurs very rarely in India (Hess, 1929). The reason for this belief is apparently the uniformly greater incidence of sunshine in this country through-



out the year. The occurrence of rickets, however, has been reported from time to time from several parts of India and in one particular region rickets is a public health problem.

Hutchison and Shah (1922) have reported a few cases of rickets in Bombay Province. In the annual reports of children's hospitals, rickets is found to be one of the diseases for which children are brought to the hospital. However, even these records do not include all the cases of rickets for the simple reason that a large number of them are recorded under fevers and respiratory diseases, the types of complaints for which the parents of a rachitic child usually seek medical advice. It is only a routine screening of the inferior radio-ulnar joints of every infant and child under five years of age going to the hospital that can truly indicate the incidence of rickets. Unfortunately such a procedure is not commonly followed even in children's hospitals. Patwardhan, Chitre and Sukhankar (1944) collected for biochemical studies 9 cases of proved rickets at one of the Bombay hospitals within a period 6 - 9 months. The occurrence of rickets as one of the major deficiency diseases of infants and children has only recently been realised in certain quarters as can be seen from a recent statement by Coelho (1950) that "in spite of our sun and the wide prevalence of breast feeding deficiency rickets is a common disease of children in Bombay".

Observations on the incidence of rickets can also be found in some of the records of nutrition surveys. In Orissa the incidence has been reported as being between 0.2 to 1.6 per cent and in Hyderabad State between 0.1 to 1.2 per cent. In a recent survey in Uttar Pradesh on 2,595 children from different districts the average incidence of rickets was found to be

3.2 per cent. In Punjab rickets occurs much more frequently. The report of the Public Health department for 1939 gives a figure of 56 per cent in 156 children of the Kangra Valley, a similar survey in Lyallpur revealed an incidence of 6.3 per cent. The figure for Kangra Valley although unusually high is more or less correct. The work of Wilson and her colleagues has shown that Kangra Valley is unique in India in having a very high incidence of rickets and osteomalacia. Wilson's studies are revealing on this subject and well worth describing.

Wilson (1931 a and b) found 400 cases of late rickets in three towns—Lahore, Simla and Amritsar. In another survey of girls in Lahore City she recorded as high a figure as 607 out of a total of 1,482 showing evidence of having suffered from rickets. According to Wilson, faulty diets, lack of sunlight (due to overcrowding in tall closely packed buildings characteristic of old Indian cities) and purdah system were responsible for this high incidence. In Ludhiana where there was no overcrowding the incidence of rickets was much lower. The criteria for diagnosis which she used in her studies were bossing of head, enlarged epiphyses of wrists and ankles, knock knee, curving of legs and irregularity in dentition. Not much difference was found in incidence in Hindu and Muslim families in one locality of Lahore, but in another there was greater incidence in Muslim girls. Wilson also examined 500 children in Srinagar (Kashmir) and found rickets prevalent in a substantial number of children. Wilson and Widdowson (1942) examined a total of 9,178 children of 5 to 15 years in Punjab, Central Provinces (Madhya Pradesh), Orissa and Hyderabad State, and found the incidence of 0.9 to 3.9 per cent in Punjab, 0.5 to 0.7 per cent in





Figure XX—A case of rickets from Punjab showing marked bony deformities.  
(*Nutrition Museum*).

Central Provinces, 0.3 to 0.7 per cent in Orissa and nil in Hyderabad. Among the Punjabis, rickets was more common in girls and of the three communities Sikhs showed the lowest incidence and Muslims the highest. These authors make a special mention of Kangra Valley where rickets and osteomalacia are rampant, although no exact figures of incidence are given. (Fig. XX)

One example of a group of 38 families is quoted; of these, 23 families were seen in which at least one member and sometimes all in the family had osteomalacia or rickets. When rachitic and nonrachitic families were grouped according to their economic status it was found that rachitic families as a whole were poorer than the groups of families which were comparatively free from the disease. The authors have examined the causes such as the type and amount of cereals in the diet, lack of vitamin D, incidence of sunshine, overcrowding and purdah system. While there is no doubt that in the rest of the Punjab the last two are operative as cause of rickets, in Kangra Valley the conditions do not differ materially from those existing in other provinces of India and yet deficiency rickets and osteomalacia are far more common in the former region. Ahmad, Sehra and Swaroop (1945) have found low calcium and high inorganic phosphorus in the serum of "normal" males and females of Kangra Valley between the ages of 5 to 64 years, the average values being 8.97 and 8.42 mg. Ca and 4.72 and 4.48 mg. inorganic P per 100 c.c. serum for males and females respectively. Among nonrachitic children too the values of calcium were 8.62 mg. per 100 c.c. serum although the inorganic phosphorus was about 4.5 mg. per 100 c.c. In rachitic children and osteomalacic women the values were



lower still as was to be expected. The estimates of calcium intakes made by Wilson and Widdowson do not justify the markedly low serum calcium values in the normal population of Kangra Valley.

Patwardhan and his associates (1944, 1945) have studied the changes in the composition of serum taking place in experimental and clinical rickets. In induced rickets in puppies they found that the changes in the concentration of serum Ca, inorganic P and total proteins were reflected in the ionic products of  $\text{CaHPO}_4$  and  $\text{Ca}_3(\text{PO}_4)_2$ . That such changes take place had been pointed out earlier by Freeman and Maclean (1941) in puppies. The observations of Patwardhan *et al* on clinical rickets (77 cases) showed that such alterations also take place in the serum of rachitic children. Dikshit and Patwardhan (1946) compared the efficacy of the increase in alkaline phosphatase activity and the unsaturation of the plasma with respect to  $\text{CaHPO}_4$  as a means of early diagnosis of rickets and reached the conclusion that the former gave an earlier indication of the presence of the rachitic process.

**Osteomalacia:** The environmental conditions which are responsible for rickets in early life probably continue to operate late as well for in Kangra Valley osteomalacia occurs widely and is responsible for much deformity of long bones of the lower extremity and the pelvis. In a series of publications Wilson (1929, 1930, 1931) has described the causation, occurrence and treatment of osteomalacia in North India, particularly in the Punjab. She has made a special study of the conditions in Kangra Valley as referred to in the last few paragraphs. Kangra is situated at an average elevation of 3,000 feet from sea level at the foot of the Himalayas. It has plenty of sunshine for





Figure XXI—A case of advanced Osteomalacia showing marked deformities affecting the spine, pelvis and lower extremities.  
(*Nutrition Museum*).

more than eight months; rainfall is restricted to the monsoon months and amounts to 100 inches per annum. The soil is deficient in lime, phosphorus and magnesium. Only one-third to one-fourth of the total area is cultivable and hence about 837 persons have to subsist per square mile. In Launa village it was found that among the poor classes 83 individuals—male and female—showed evidence of early and late rickets or osteomalacia. Among 47 persons of the landowner families, 16 individuals were affected. Thus there is little doubt that poverty of the soil in lime and phosphorus together with the diets excessively rich in cereals may be responsible for predisposition to rickets and osteomalacia. The latter condition progressively gets worse with each successive pregnancy and causes extreme softening and bending of bones (Fig. XXI).

That osteomalacia is not confined to Punjab can be seen from a few reports appearing from other parts of India. Green-Armytage (1928) mentions that in Calcutta osteomalacia appeared to be common among Marwari (money-lenders and businessmen) community and not uncommon among the purdah observing Muslims. Out of 2,870 maternity cases seen in Eden Hospital, Calcutta, in the course of 2½ years, 26 craniotomies and 15 caesarian sections had to be performed for osteomalacic conditions alone. Wills (1931) has reported on 20 cases in pregnant and non-pregnant women in Bombay City. Heilig (1943) comments on the occurrence of osteomalacia in Mysore among women of child bearing age belonging to the community which observes purdah system. There is no doubt, however, that the condition is much less common in the south than in the north. Table XXIII prepared from data collected in 1935 can be considered to be illustrative.

TABLE XXIII—INCIDENCE OF CONTRACTED PELVIS  
IN SOME MATERNITY HOSPITALS IN INDIA

Hospital	Contracted Pelvis
Lady Hardinge Hospital, New Delhi	1 in 11 deliveries.
Maternity Hospital, Agra, U.P.	1 in 15     „
Cama Hospital, Bombay	1 in 35     „
Caste and Gosha Hospital, Madras	1 in 99     „

## NUTRITIONAL OEDEMA

In comparison with the incidence of other nutritional disorders, simple nutritional oedema is less rarely seen. Its association with nutritional diarrhoeas in adults and infants and children has been described earlier. A few other cases have been reported periodically from different parts of the country. It is quite probable that the larger hospitals may have recorded cases of nutritional oedema, but published reports are comparatively few. Puttaiya (1944) reported its occurrence in certain women patients who responded to high protein diets. Walters, Rossiter and Lehmann (1947) observed it in a very small proportion of 2,000 Indian prisoners of war in the East. Datta (1947) studied the total serum protein and its fractions in 30 cases of nutritional oedema in Bombay, 18 of his cases had total protein below 5 gm. per 100 c.c. serum; in the remaining 12 the values were between 5 gm. and 6 gm. per 100 c.c. The serum albumin in 25 cases was below 2.5 gm. per



100 c.c. Recently, Gopalan (1950) investigated 12 cases of nutritional oedema in Madras. He found that the urines of these patients had a larger concentration of anti-diuretic hormone as compared with that in normal urine. Gopalan is of the opinion that the decrease of the serum proteins, i.e., albumin, is not the only factor responsible for nutritional oedema, probably there is a greater amount of the anti-diuretic hormone circulating in the blood which depresses the excretion of urine and aids in the escape of fluid in the extravascular spaces.

Hardikar and Rao (1943) and Rao (1946) describe cases in which the evidence points to a defective diet being the sole cause of oedema and ascites. Rao gives an account of 52 cases of nutritional ascites in children, adolescents and adults of both sexes. The presence of ascites, together with oedema of the legs and trunk (in some cases even the face) was the feature of the disease. Anaemia of varying degree, commonly hypochromic and normocytic or slightly macrocytic was present. Serum proteins which were done only in eight cases showed albumin values between 1.31 to 2.43 gm. per 100 c.c. Mild signs of beri-beri were present in 14 and in 2 they were severe; evidence of deficiency of riboflavin and nicotinic acid was found in only one. Other investigations such as examination of ascitic fluid, liver and renal function tests were done, though unfortunately only in a very small number of cases, hence it is difficult to interpret the findings. The remarkable part of the report is that which refers to the response to treatment with good hospital diet starting with bread and milk and gradually changing over to a complete diet. The average period of hospitalisation for 32 cases which completely recovered was 37 days.

Rao reviews the earlier literature on the subject but fails to find clearcut evidence of the occurrence of similar conditions in other parts of India. He refers to a few reports from Lucknow and Calcutta between 1920 - 30 in which the circumstantial evidence was in favour of ascites being of nutritional origin. In times of famine and starvation, one is likely to meet with cases of nutritional oedema and the investigations carried out in Calcutta indicate that cases with oedema alone and oedema with ascites were found among chronically malnourished people suffering the added misery of starvation of Bengal famine. Mention of a few such instances is made in a report (1944) on the treatment of destitutes and starvation cases in Bengal by Bose, De and Mukherjee (1946).

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## CHAPTER XIX

### DEFICIENCY DISEASES (CONTINUED)

#### ENDEMIC GOITRE

SIMPLE goitre is endemic in the Himalayas and the sub-Himalayan region. The endemic region extends from the northwest frontier along the northern boundary of India to the northeast border in Assam. There are no known endemic foci in the rest of the country. In certain regions within the endemic area the incidence may be unusually high. McCarrison (1921) mentions that in some villages in Chitral and Gilgit on the northwest frontier no person was free from goitre; in the village of Awi, he records that about 60 per cent of the children were born with congenital goitre, and many were cretins. McCarrison (1926) also records 50 per cent incidence of goitre in children of the Lawrence Military School, Sanwar, in the Simla Hills, Punjab. The incidence increased to 80 per cent when the children stayed there for a few years. In a survey of Kangra Valley an incidence of 34 per cent has been recorded (Punjab Public Health Department Report 1939). Stott *et al* (1931) have studied the distribution of goitre in Himalayan, sub-Himalayan, foothill and low marshy regions of the United Provinces where they record an incidence of 30 per cent to 50 per cent. As is usual with endemic goitre the occurrence of cretinism and deaf-mutism is seen to a remarkable degree in these regions (Fig. XXII).

Stott *et al* have examined the 1911 and 1921 census figures for the occurrence of congenital





Figure XXII—A case of goitre and a cretin from the goitrous regions of the (United Provinces) Uttar Pradesh.

*(By courtesy of the Indian Journal of Medical Research).*



deaf-mutism in India. They find a larger incidence per 100,000 population in the regions where goitre is endemic as the following figures based on 1921 census will show:

State or Province	Kashmir	Punjab	Bombay	Madras	Central India	Hyderabad
Number of deaf-mutes per 100,000	138	89	55	51	34	27

Even within the same province the proportion of deaf-mutes in the goitrous regions was very much higher than in non-goitrous parts as shown in Table XXIV for three provinces abutting on the Himalayas.

TABLE XXIV—DEAF-MUTE RATES IN GOITROUS REGIONS AS COMPARED TO PROVINCIAL AVERAGE RATE

State	Area	Deaf-mutes per 100,000
Punjab	...	...
	Himalayan Division	257
	Provincial average	89
Bihar	...	...
	Champaran District	169
	Provincial average	72
Assam	...	...
	Naga Hills	490
	Provincial average	70

Thus in India too as elsewhere deaf-mutism like cretinism is associated with endemic goitre.

Although it has been known for a long time that deficient iodine intake is the primary cause of simple goitre, McCarrison held other views. His careful

study at Gilgit suggested to him that probably the use of polluted water had some relation to incidence of goitre. This opinion received further confirmation by his observations at the Lawrence Military School, Simla Hills. As mentioned earlier the school showed an incidence of 50 per cent to 80 per cent among its boarders. It had a local unprotected water supply which was suspected to be polluted. In 1918 a new pipe line was laid from Kasauli, a few miles distant from Sanwar. Within a few years goitre in the school became rare. In 1926, it was recorded by the Medical Officer of the school that "One year's experience has convinced me that goitre is now entirely absent". McCarrison was of the opinion that bacteriologically pure supply of water was responsible for this change. He had the soil and water at Sanwar and Kasauli analysed in three different laboratories. The iodine content of water—new and old supplies—varied between traces to 0.1 p.p.m., and that of the hill side soil varied between 1.0 to 4.5 p.p.m. Essentially, however, there was no difference between the soils of Sanwar and Kasauli. It must also be mentioned that so far as could be ascertained there had been no changes of any importance in the diets of the school boarders. It was indeed surprising that in a locality where goitre was known to be endemic for over 70 years, the disease should be almost completely eradicated within a space of eight years.

This remarkable observation was followed up by further experimental work in the laboratory. McCarrison, Newcomb, Viswanath and Norris (1927) analysed the soils from endemic and other zones. Although the incidence of goitre in Chitral and Gilgit varied from village to village from 10 to 65 per cent, the iodine content of soil in the affected and goitre free

areas was less than 1 p.p.m. McCarrison, Sankaran and Madhava (1931) analysed the urines of 36 goitrous and 33 nongoitrous individuals from Gilgit. These urines were collected over potassium carbonate and sent to Coonoor which is nearly 2,000 miles away. It had been ascertained that urines stored over  $K_2CO_3$  did not lose their iodine. The iodine content of goitrous urine varied from 4  $\gamma$  to 18.0  $\gamma$  per litre with an average of 7.2  $\gamma$ , that of nongoitrous urine had a range of 3  $\gamma$  to 22.0  $\gamma$  with an average of 9.2  $\gamma$  per litre. Thus there appeared to be no significant difference between the urinary excretion of iodine in normal subjects and those with goitre. McCarrison and Sankaran (1931) confirmed their earlier findings by analysing another batch of urines from fresh subjects.

McCarrison and his colleagues then turned their attention to the production of goitre in animals. McCarrison (1930) found that when rats were fed diets deficient in vitamins A and D, with high or low protein content, the thyroids were enlarged. He also observed that if rats were kept in cages which were only infrequently cleaned and hence had accumulated collections of stale food, excreta, etc., thyroid enlargement resulted, whereas in controls fed the same diet, but kept in cages cleaned daily and kept scrupulously clean, the thyroids were normal. In another experiment, iodized water (containing 0.1 mg. iodine per litre) was supplied to two batches of animals, fed and maintained under conditions described above. Rats kept under insanitary conditions again showed a significant enlargement of thyroid as compared to controls. McCarrison observed that the provision of additional iodine through drinking water prevented the thyroid enlargement induced in rats living under



unhygienic conditions. On the basis of further experiments, McCarrison (1933) formed the opinion that due to bacterial action a goitrogenic agent arises which can be waterborne and which can cause enlargement of thyroid on continued ingestion. McCarrison (1931 and 1933) also studied the goitrogenic action of diets containing cabbage, soya bean and groundnut respectively. In diets containing sufficient amounts of added potassium iodide, but deficient in various other nutrients, soya bean and groundnut caused marked enlargement of thyroids. These observations were later confirmed in the case of soya bean by Sharpless (1938) and Wilgus *et al* (1941) and for cabbage by Hercus and Purves (1936) and Kennedy and Purves (1941).

McCarrison has summarised his own views relating to the causation of endemic goitre in a memoir published in association with Madhava in 1932. He is of the opinion that while deficiency of iodine in soil and water is frequently associated with endemic goitre in Himalayan region, this association is not invariable. Other factors, such as defective dietaries and unprotected bacteriologically impure water supplies, or the presence of goitrogens in certain foodstuffs are other contributory factors. He does not believe that chemical composition of water has any relation to goitre. Yet, there are certain observations on record which indicate the relationship of hard waters with goitre. Stott *et al* (1931) and Stott (1932) have commented upon this association which was very marked in goitre regions of the United Provinces. Stott (1935) further refers to the work of Turton in Derbyshire where also the association between lime rich water and goitre incidence was noted. More recently Parra (1948) has reported similar findings in Colombia,

hence it must be admitted that the almost similar findings reported from three different countries can hardly be the result of fortuitous circumstances. The association between hardness of waters and endemic goitre obviously requires further investigation.

From a consideration of all the observations described above, the conclusion emerges that there must be more than one aetiological factor in the production of simple goitre, the principal of which is, of course, deficiency of iodine. It may be possible that some of the conditions leading to the hypertrophy of the gland may operate by interference with the utilization of iodine or by increasing the demands on iodine so that a higher level has to be ingested to overcome these effects. The well known experiments of Marine and Kimball (1921) have, however, demonstrated the value of iodine prophylaxis. In India where a wide field for such work exists, there has unfortunately been no attempts to find how iodine prophylaxis would work. It is imperative, both from the scientific as well as public health standpoint that trials with iodized salts should be undertaken, for these, in the opinion of the FAO/WHO Expert Committee on Nutrition (1950), appear to be the best and cheapest way of not only preventing the disease, but incidentally of obtaining valuable information on the aetiology of simple goitre.

### NUTRITIONAL OEDEMA SYNDROME

The syndrome 'Malignant Malnutrition' also known as 'Kwashiorkor', has been described from various parts of the world. The first descriptions of the syndrome appeared from the African continent where Williams (1933) gave it the name of 'Kwashiorkor'. Later several reports dealing with its occur-

rence appeared from Mexico, Brazil, Cuba, Ceylon, etc. It is usually the children in the post-weaning period who suffer from the disease, although occasionally it is seen in adults as well. The characteristics of the disease are diarrhoea, oedema, dermatosis and a fatty liver. Among the dark African races depigmentation of hair and skin may be seen. High mortality has been a feature reported upon by several workers. Recently Trowell (1949) has summarised the existing knowledge on the occurrence, etiology, prognosis and sequelae of Kwashiorkor.

The appearance of the disease in India was first reported by Hare (1947) who described four cases with the characteristic features mentioned above. All the four cases terminated fatally. Chaudhuri (1947), Passmore (1947) and Raman (1948) also make references to the occurrence of the disease in India. Later, Ramalingaswami, Menon and Venkatachalam (1948) gave under the name infantile pellagra an account of five cases seen at Coonoor. They mention, however, that the condition was more akin to 'Malignant Malnutrition' than to infantile pellagra. All their cases also had a fatal ending. Recent experience with a larger number of cases investigated at Coonoor shows that the prognosis is not so bad as the fate of the earlier cases had led one to believe. Achar (1950) has treated 78 cases in Madras. He suggests that the term nutritional dystrophy would be more apt than either Kwashiorkor or Malignant Malnutrition. The name Kwashiorkor is of African derivation meaning a 'red boy' and originated in the fact that red discolouration of hair and depigmentation of the skin were observed in children suffering from the disease. It appears, however, that in patients from races more lightly coloured than the Africans, red discolouration



A

Figure XXIII — Cases of nutritional oedema syndrome in children.



B



C



D

Figure XXIII—(Continued) The characteristic apathetic attitude is seen in the child in picture B. Note the well marked crazy pavement dermatosis affecting particularly the flexures and pressure sites.—(Nutrition Museum).



of hair may not always be seen although the other characteristic features of the syndrome are present. Naturally, the term is not strictly applicable in such cases. The term malignant malnutrition is also inapt since a better understanding of the aetiology of the disease and rational treatment have been effective in considerably reducing the mortality. On the other hand the terms "nutritional dystrophy" of Achar and "chronic subnutrition" of Trowell appear to be diffuse in their meaning and capable of application to several syndromes arising out of subacute or chronic nutritional deficiencies. Oedema is a central and minimal feature of the syndrome under discussion; other characteristics, such as diarrhoea, palpable liver, dermatosis, anaemia and depigmentation of hair may individually or collectively accompany the oedema. These facts distinguish it from the simple nutritional oedema. Our colleague, Dr. C. Gopalan has suggested the name 'Nutritional Oedema Syndrome' and we feel that under the present circumstances it is the most suitable of all for the syndrome under discussion (Fig. XXIII).

The experience of several workers abroad as well as of those in India has been that every patient does not show all the characteristic features of the syndrome, the constant features being restricted to arrest of growth, diarrhoea, oedema, and anaemia of varying degree. Anatomically the liver may or may not be enlarged to a palpable degree. It is probable, however, that even in livers that are unpalpable, fatty infiltration may have taken place. Some patients present themselves with crazy pavement dermatosis, some develop it during treatment and some do not. Alopecia and/or depigmentation of hair has been seen in some cases. A certain proportion of the patients

shows evidence of suffering from dysenteric disorders, some others have a history of having so suffered, while there is quite a large number in whom no evidence of dysentery past or present can be elicited. In view of this it does not appear as if dysentery has a primary aetiological role in the disease; it may merely accentuate the effect of chronic malnutrition. Infestation with ascaris is another factor to be taken into consideration. In four cases reported by Hare, ascaris ova were found in the stools of one patient only. Ramalingaswami *et al* found no helminth infestation in their cases although in a later series of 63 cases investigated at Coonoor 25 were so infested. It must be mentioned that in these cases only a single stool examination was done and no concentration method was used. Too much stress, however, cannot be laid on a single examination of stools. Patients under treatment not showing ascaris ova in stools have been found to pass adult worms during the period of hospitalization. It is probable, therefore, that ascaris infestation is more common in these patients than the above figures would lead one to believe. In fact, Achar has reported that 70 patients out of 78 of his series harboured round worms. Among the backward people of the tropical regions living under primitive hygienic conditions, helminth infestation is fairly common, and children are particularly susceptible to ascaris infestation. It will be worthwhile, therefore, to study carefully the role of round worms in the evolution of the syndrome. Trowell (1949) has discussed the combined role of tropical disease and chronic subnutrition in the development of the full clinical picture of nutritional oedema syndrome. It is quite possible, as suggested by him, that ineffectively treated malaria, dysentery,

hook-worm or ascaris infestation, etc., may accelerate the processes already set in motion in a chronically under-and malnourished child. It has to be remembered that already these children, like so many others of their class, have had a bad start in life on account of the state of chronic subnutrition present in the mothers. The patients are mostly derived from poor and very poor class of people and neither the adults nor children can be said to be living on diets which are qualitatively or quantitatively adequate. Superimposed on this are the meagre and faulty diets on which the children are fed after weaning. These considerations provide ample reason to believe that first and foremost, chronic malnutrition is the basis of this syndrome, tropical disease playing a secondary but an important role in its development. Trowell's (1949) conception of different factors operating in the development of marasmic malnutrition and malignant malnutrition is attractive as a working hypothesis. However, one will have to collect more evidence before its correctness or otherwise can be proved.

As a rule these infants are breastfed for the simple reason that the family cannot afford cow's milk. Secondly as it grows older the infant does not receive satisfaction at the breast and is given rice water (kanjee) as one or more supplementary feeds. Later when the child is weaned, it is put on to solid food consisting mainly of rice with very little of other nutritious foodstuffs. This regimen leads first to the arrest of growth and the appearance of deficiency symptoms, such as night blindness, xerosis of scleral conjunctiva or cornea or both, anorexia, angular stomatitis, cheilosis and what is more important, the onset of diarrhoea. It is this latter complication with

possible intestinal infection or helminth infestation that leads to a restriction of food and worsening of the situation. The result ultimately is the development of a multiple deficiency in which the predominant feature is the deficiency of protein.

That the above concept is not far from correct can be seen from the evidence converging from two angles, firstly, the recognition by Waterlow (1948) and by Altmann (1948) that the disease was similar to that described as 'Mehlnahrschaden' of the old German pediatricians seen in Central Europe in babies fed carbohydrate rich and protein poor foods, and secondly the observation made by Waterlow (*loc. cit.*), Russell (1946) and Altmann (1948) that milk is of great benefit in the treatment of this syndrome. The observations of Veghelyi (1948) in Budapest revealed that on low protein diets the digestive juices secreted by the pancreas decreased even before changes in blood and liver could be demonstrated. Diarrhoea set in later. Waterlow (*loc. cit.*), and Davies (1948) found atrophic changes in the acini of the pancreas which may have been the result of protein deficiency.

The treatment of the disease has undergone changes as its aetiology has become clearer. The lesions accompanying the syndrome or forming part of it gave an impression at the beginning that the condition was due to a deficiency of one or more members of the vitamin B complex. Thus Trowell (1940) believed that it was due to a deficiency of nicotinic acid, but later Trowell and Muwazi (1945) revised this opinion in favour of a multiple deficiency. Hughes (1946) believed that riboflavin deficiency was the predominant feature in a complex deficiency manifested as Kwashiorkor; cases showing evidence of beri-beri accompanying the nutritional oedema







Figure XXIV—Nutritional oedema syndrome: Effect of high protein diet—  
(left) patient on admission, (right) same child after six weeks  
of treatment with skim milk supplement.

*(Nutrition Museum).*

syndrome have also been not infrequently encountered. In spite of the above, however, the response to vitamin therapy has been disappointing. On the other hand Gillman *et al* (1944) and Gillman and Gillman (1945) and Trowell (1946) obtained good results with desiccated stomach, which Gelfand (1946) was unable to confirm in his cases. Experiences at Coonoor and of Hare in Assam too have been unsatisfactory with vitamins and in the former instance also with desiccated stomach. Menon (1950) has observed in Coonoor that simple treatment with reconstituted skim milk in uncomplicated cases gave the most encouraging results. At the commencement, two ounces of the powder reconstituted by addition of eight volumes of water was given in divided doses. The dose of skim milk was gradually increased as diarrhoea was controlled and the general condition improved. Gradually other solid foods were introduced. The treatment lasted for 17 to 43 days. The cases which showed associated ocular symptoms of vitamin A deficiency were given vitamin A concentrate in addition. It is noteworthy that when diarrhoea lessened and stopped, oedema gradually receded and the dermatosis and angular stomatitis, cheilosis, glossitis etc., also responded. In ten cases with diarrhoea, oedema and dermatosis treated on this regime, seven were discharged cured. In twenty cases with diarrhoea and oedema but without dermatosis, sixteen were cured. In earlier cases where mixtures of nicotinic acid and riboflavin were given dermatosis and angular stomatitis responded, but diarrhoea did not and the patients died (Fig. XXIV).

Achar (1950) has developed a routine technique of blood transfusion and/or concentrated human serum transfusion, together with acidified half cream

milk fortified by casein products given orally. The reason for resorting to transfusion is given as (a) the low haemoglobin, (b) obvious hypoproteinaemia and (c) the inability of the liver to synthesize serum albumin. Achar reports a dramatic change in the child after two weeks. Trowell (1949) also mentions the use of blood transfusion in the most feeble cases and it must be admitted that in such cases it may indeed be necessary. It is to be doubted, however, that the failure of intestinal function in a majority of cases has so far advanced as to make parenteral therapy a routine therapeutic procedure of choice. Further, additional evidence will have to be produced to prove the suggested lack of capacity on the part of the liver of these patients to synthesize proteins such as plasma albumin. The experience at Coonoor with about 63 cases treated till now indicates that in a majority of cases the intestines and liver can respond to oral treatment. The diarrhoea, unless it is caused by infection, can be controlled by skim milk alone. Cow's whole milk is unsuitable as it may not be tolerated in the early stages. The diarrhoea, as has also been observed by workers at Coonoor, is fatty showing discrete fat globules. With skim milk the fat globules gradually disappeared from the stools as the diarrhoea lessened. Once the stools are well formed larger quantities of skim milk can be given together with other solid foods. With this regimen hypoproteinaemia has been found to respond quite well as was shown by an average value of 7.12 gm. total protein per 100 c.c. plasma in 14 cases after treatment as against 3.86 gm. per 100 c.c. in 16 patients before treatment.

Recently, Gopalan and Venkatachalam (1950) have commenced treating patients with testosterone

propionate in addition to skim milk. The rationale of this therapy is the well known effect of the androgen on nitrogen retention and utilization, both in animals and in human beings (Kochakian, 1946). The effect is seen to better advantage in debilitated subjects. The number of cases of nutritional oedema syndrome tried with the androgen is small, as yet only eight cases have been treated. The results, when compared with those obtained with controls on skim milk alone are distinctly encouraging. The disappearance of oedema was quicker in testosterone treated group than in the controls. There was, however, little difference in the time taken by plasma proteins to reach normal values. These observations offer a promising new line of treatment and further experiments will be watched with interest.

A reference to the work pointing to pancreatic dysfunction has been made above. Veghelyi *et al* (1950) have reported that in protein deficiency the (external) secretory activity of pancreas suffers owing to certain degenerative changes seen in the acini. This must result in the incomplete digestion of food and its consequent faulty absorption. Veghelyi (1948) had found that the concentration of some pancreatic enzymes in his patients' serum was low, being restored to normal after recovery. Our experience has been that in nutritional oedema syndrome, plasma lipase and esterase activities are low. The results of some observations are given in Table XXV. Plasma esterase was estimated by the method of Cherry and Crandal (1932) and lipase by that of Goldstein *et al* (1948).

TABLE XXV  
TOTAL PROTEIN AND LIPASE AND ESTERASE  
ACTIVITY OF PLASMA IN NUTRITIONAL  
OEDEMA SYNDROME

	No.	Before Treatment	No.	After Treatment
Total plasma protein				
Range gm. %	16	3.55 to 5.55	14	6.10 to 8.70
Average „	...	3.86		7.12
Plasma Esterase Units/100 c.c.				
Range	14	30 to 205	14	100 to 203
Average	...	83.7		150.6
Plasma lipase Units/100 c.c.				
Range	7	20 to 65	10	100 to 160
Average	...	35.0		136.5

Seligman and Nachlass (1950) have expressed doubts regarding the specificity of the current methods of plasma lipase estimations. Subsequent work at the Nutrition Research Laboratories has confirmed these doubts. However, it has now been possible to develop a method for lipase estimation in plasma by taking advantage of the complete inhibition of plasma esterase action on tributyrin in the



presence of sodium tauroglycocholate which at the same time exerts an activator effect on lipase. By this method, the presence of lipase in plasma has been satisfactorily demonstrated. The values given in Table XXV, however, are those obtained by the older methods and hence should be interpreted with caution.

Low plasma esterase values suggest a deficient liver function. Apart from the abnormal histological picture presented by the livers in nutritional oedema syndrome, Holmes and Trowell (1948) have demonstrated the failure of liver cells (from biopsy specimens) to store glycogen in presence of liberal amounts of glucose. Waterlow (1950) has found lowered choline esterase activity of biopsy specimens of liver in two cases. Histologically the sections from biopsies before treatment showed shrunken cells with a higher nuclear count per standard field than in specimens obtained after treatment. At this time the cells were normal in appearance, nuclear count lower and choline esterase activity higher. It would be interesting to continue studies in other directions for comparing the results of biochemical examination with the histological appearance of biopsy specimens obtained serially in the course of treatment.

Hepatic structure in nutritional oedema syndrome has been studied by workers at the Nutrition Research Laboratories in over thirty cases with the help of aspiration biopsy technique. In more than half the cases, the biopsy was repeated after a period varying from 4 to 8 weeks of treatment. At the time of first biopsy, moderate to severe fatty infiltration of liver cells was seen in almost every case as has been reported in the African and West Indian cases. The fat was present both in the form of regal of globules

and small droplets lined by several nuclei probably due to coalescence of fat globules in adjacent liver cells as described by Hartroft (1951). Contrary to common belief, appreciable amounts of glycogen displaced towards the periphery of the cells could be demonstrated in many cases. Some reduction in the number and size of pyroninophilic bodies in the liver cells was also observed in the majority of cases. In livers with severe fatty infiltration neither pyroninophilic bodies nor glycogen could be demonstrated in appreciable amounts; there seemed to be no room for these components in the liver cells which were distended with fat. No necrotic changes were seen in any of the sections and there was no pigment either in the liver cells or Kupffer's cells. The mesenchymal change consisted of a mild histiocytic infiltration of vascular tracts, with no definite increase or thickening of reticular fibres. The Kupffer's cells in many cases showed proliferative changes and several polymorphonuclear cells were seen in the sinusoids.

Biopsy specimens taken after 4 to 8 weeks of treatment with skimmed milk powder, showed in most cases a remarkable regression of fat and a concurrent increase of glycogen and ribonucleoprotein particles in the liver cells. The smaller fat droplets tended to disappear first, but the larger coalesced masses of fat persisted even after 8 weeks of treatment. Some of them, however, failed to stain completely with Sudan IV, suggesting a reduction in their fat content. With the retreat of fat, there was increased cellular infiltration and Kupffer's cell hyperplasia, but no definite fibrosis was observed. The absence of fibrosis is probably due to the short periods after which the biopsy was repeated. However, in a child who was followed up for one year after being discharged

cured' and was readmitted with recurrence of the syndrome, well-defined diffuse fibrosis, bile duct hyperplasia and moderate fatty change were observed.

In the course of this work, Ramalingaswami, *et al* (1952) found that there was an abnormal accumulation of cholesterol in the livers of certain cases of nutritional oedema syndrome. Their attention was first drawn to this by the detection of crystalline structures in the form of rhombic plates in the fat globules in the liver cells. The crystalline material was absent in sections from the same livers which were exposed to the action of alcohol, acetone and xylene, indicating that it was a lipoid substance. It was later found to be doubly refractile in polarized light, which suggested that it might be cholesterol. On subjecting the biopsy specimens, fixed in neutral formalin, to chemical estimation of cholesterol by the Liebermann-Burchard reaction, abnormally high values for cholesterol (in two cases, as high as 10 per cent by weight of dry liver tissue) were obtained. Although only a small number of cases have been examined so far, the presence of excessive amounts of cholesterol in nutritional oedema syndrome must be considered a significant finding. The pathogenesis of cholesterol accumulation in nutritional oedema syndrome is being investigated.

The possible after-effects of nutritional oedema syndrome in cases which recover have been discussed by Trowell (1949). In India, unfortunately, there is little information on this aspect particularly as the interest in this disease has been evinced only recently. Portal cirrhosis reported as one of the sequels is not uncommon in India among the poorer classes in the fourth and fifth decades of their life. If the evidence from Africa has any bearing on the role of

malnutrition in the aetiology of liver disease, it is quite probable that similar causes may be operative in this country too. It is better, however, not to venture far in the realm of speculation in absence of much evidence on the subject. On the other hand, it is safe to observe that infantile biliary cirrhosis seen in certain parts of India is not one of the sequelae of the nutritional oedema syndrome.

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## CHAPTER XX

### OTHER DISEASES ASSOCIATED WITH DIETARY HABITS

#### URINARY CALCULI

IN HIS search for the known and unknown manifestations of the influence of deficient or imbalanced dietaries on the body organs, McCarrison (1921) noted the effect of scorbutic diets on the bladder of guinea pigs. This was seen as congestion of the mucous membrane and patchy ecchymoses at various points in the mucosa. Histologically, haemorrhagic infiltration of all coats of the wall of the bladder and degenerative changes in the epithelium of the mucous membrane were found. McCarrison believed that such changes could occur in human subjects in vitamin C deficiency and might explain haematuria in human scurvy. It is quite possible that inflammatory or degenerative changes in the bladder may provide a nidus for the formation of vesical calculi. Vitamin C deficiency, however, has not been associated with urinary calculus. However, McCarrison had made a note of the observation of Osborne and Mendel (1917) that there may be a possible connection between partial or complete dietary deficiency of vitamin A and the occurrence of phosphatic urinary calculi. He (1931) collected information from various hospitals in the country on the incidence of urinary lithiasis and the dietary history of the patients. It is true that one cannot consider the hospital statistics in India as a reliable index of the incidence of a disease among the general population. However, the data collected by McCarrison have given some interesting

information and revealed trends which he was able to follow up in the laboratory. The incidence of "stone" as ascertained by McCarrison is illustrated in Figure XV. The incidence of stone was found to vary enormously, being highest in Sind, North-West Frontier Province and Punjab. It decreases as one proceeds from the north towards the east or south, the only exception being Manipur on the eastern border where again the incidence is high.

The incidence of urinary calculi did not appear to be related to physical features of the country. On the other hand it would seem as if the disease was more common in parts with extremes of heat and cold and great disparity between night and day temperatures and less so in the equable regions of the south. Its incidence appeared to be associated with diets based on wheat, with one exception, however, and that in the case of Manipur where rice forms the staple cereal. Guided by these considerations McCarrison undertook an extensive series of investigations on the experimental production of calculi in albino rats.

McCarrison (1927, 1930) succeeded in producing vesical calculi in a certain proportion of rats by feeding them diets consisting of a cereal 53 parts, linseed meal 20 parts, corn flour 25 parts, NaCl 1 part and  $\text{Ca}_3(\text{PO}_4)_2$  1 part. Among the cereals he found whole wheat to possess the highest calculogenic property, for the relative incidence on wheat, millet, rice and bajra was 31.7 per cent, 7.7 per cent, 9.1 per cent and nil respectively. Milk was shown to exert a protective action. The "stone promoting factor" in whole wheat was not found in white flour. McCarrison observed that the addition of 0.32 gm. CaO to 20 gm. of a ration made up of 97 parts white bread

and 3 parts dried yeast increased the incidence of stone among his animals from 16.6 to 42.8 per cent. The above diet was deficient in vitamins A, C and D and contained excess of calcium. McCarrison, therefore, suggested that a deficiency of vitamin A contributed to the formation of urinary calculi and that conditions for stone formation were more favourable when there was excess of calcium in the diet. In a later publication McCarrison summarised the results of his experiments. He points out that whereas in 416 rats on complete synthetic diets and 428 rats on stock colony diets there was not a single case of stone, there were 205 cases of stone in 844 faultily fed rats. The main defects in the diets of the latter group were deficiencies of vitamins A and D and an imbalance of calcium and phosphorus. Calculi were found in the bladder, ureters and kidneys. The calculus deposits in the bladder on "lime diets" were made of numerous small and large rounded, nonfacetted and non-pigmented grains or pellets weighing from a few mg. to 2,085 mg. In the ureter there were only impacted granules.

Newcomb (1929) analysed 100 human vesical calculi collected from all over India. Of these 9 were pure urate, 2 pure phosphate and 3 pure oxalate stones, the remainder being mixtures of urate-phosphate, urate-oxalate and phosphate-oxalate stones, the most common being phosphate-oxalate stones (38). In 25 stones all the three salts were present. The average mineral composition of these stones gave 26.4 per cent ash of which over 94 per cent was accounted for by phosphates and sulphates of calcium and magnesium. Newcomb and Ranganathan (1930) analysed a further batch of 121 human vesical calculi of which the average composition was  $\text{CaC}_2\text{O}_4$  24.9 per cent,  $\text{Ca}_3(\text{PO}_4)_2$  7.4 per cent,  $\text{MgNH}_4\text{PO}_4$  6.9 per cent,

protein 6.2 per cent, uric acid 49.7 per cent and undetermined 4.9 per cent. There were large variations in the series but in 84.6 per cent of stones, uric acid or urates was present which agrees well with the figure of 83 per cent observed by Newcomb in his earlier series. Ranganathan (1930) and Newcomb and Ranganathan (1930) also analysed the urinary calculi produced by McCarrison in rats kept on calculogenic diets. In all, 100 calculi were analysed. Their weights varied from 2 mg. to 800 mg., and the stones were of two types, (a) calcium stones where calcium was present as carbonate or hydroxide and (b) magnesium ammonium phosphate stones. Diets containing excess of calcium gave rise mainly to calcium stones. There was no uric acid or urate in rat stones as was to be expected since rat is capable of metabolising uric acid while apparently the human being is not. Ranganathan (1931) later analysed 21 vesical calculi obtained from cattle; these stones were small, rarely exceeding in size a small pea, and were multiple, often occurring up to 100 or more in a single animal. A comparison of the average composition of stones from these three species of mammals given in Table XXVI is interesting.

It will be clear from the table that urinary calculi can result from faulty diets. The composition of natural human calculi markedly differs from that of experimentally produced calculi in rats. The latter approximates more the stones obtained from cattle. Human stones contain varying amounts of uric acid and urates; cattle and rat stones do not. Then again considerable amounts of oxalate are present in human stones; in rat and cattle stones its amount is extremely small or it may be even completely absent. It must be admitted that although the role of dietary



TABLE XXVI—COMPOSITION OF VESICAL CALCULI

Per 100 gm. of air dry material							
Stone	Mois- ture	Total N	P <sub>2</sub> O <sub>5</sub>	CaO	MgO	C <sub>2</sub> O <sub>4</sub>	CO <sub>2</sub>
Human	7.2	17.9	6.9	14.9	2.0	14.0	—
Cattle	3.1	0.4	0.9	44.0	4.8	0.6	39.1
Rat	37.8	8.3	33.1	1.1	13.8	0.3	— (Phos- phate stone)
Rat	14.2	1.4	1.1	37.7	1.0	—	21.0 (Cal- cium stone)

defects in the predilection to stone formation in human beings cannot be altogether ignored the evidence presented above is largely circumstantial and additional proof is still necessary. Further it must be admitted that the type of diets which McCarrison used for inducing stone formation in rats with greater frequency is hardly comparable with the diets obtaining in regions where clinical cases of urinary calculi are fairly common. Megaw (1933) analysed the hospital statistics from various districts in Punjab and found that the incidence of stone in relation to total hospital admissions in the three principal communities in Punjab worked out as follows :

Community	Incidence %
Hindus	2.08
Muslims	3.62
Sikhs	1.05

Megaw felt that although the general dietary pattern was the same, Sikhs consumed more milk and

hence had a better balanced diet than the other two communities. This probably was responsible for the comparatively lower incidence among them.

Wilson and Mukherjee (1935) state that one possible reason for the calculogenic property of wheat diet is that the amount of urine excreted in 24 hours is smaller than on rice diet. In one subject, where the cereal in the diet was the only variable, they observed that the average urine volume on wheat diet was 1,578 c.c. as compared to 2,954 c.c. on rice diets. The pH of urine was almost identical. The oxalate content of wheat diet was about five times that of rice diet. The authors, therefore, suggest that owing to a smaller urine volume the less soluble constituents tend to precipitate, forming a nucleus for the future stone. They do not believe that keratinisation of vesical epithelium is ordinarily responsible for stone formation. The observations of Wilson and Mukherjee carried out on one subject only are suggestive and do merit some consideration; confirmation in a large number of subjects would of course be necessary.

In 1947 a fresh attempt was made to collect information on the incidence of urinary calculi from different regions of India. The procedure adopted was similar to that followed by McCarrison in his investigation in 1928. Owing to unsettled conditions prevailing in India in 1947 the information was not forthcoming on as extensive a scale as in 1928. It was possible, however, to compare the returns for some of the regions from which it appeared that, in the main, the general picture of incidence had not appreciably altered over the last twenty years.

## EPIDEMIC DROPSY

Epidemic Dropsy was first reported in Calcutta in 1877 and since then numerous outbreaks, in epidemic form, have occurred in Bengal, Assam, Bihar and as far west as the Uttar Pradesh. Banerjee (1928) reported an outbreak in Allahabad, Sagayam (1927) among Indians in Fiji Islands, Dalal (1929) among the Bengali community in Rangoon and Kamath (1928) in Ganjam District of the Northern Circars. A closer scrutiny of these reports shows that with the exception of the last mentioned outbreak the disease has occurred among Bengalees whether they lived in Bengal or in other parts of the world, provided, of course, they carried their dietary habits with them.

The reasons for including epidemic dropsy in this book are that (1) its occurrence is connected with food although not with nutritional deficiency and (2) at one time it was considered to be identical with a nutritional disease, viz., the wet type of beri-beri. Although the latter concept no longer holds the field it is certainly interesting to relate how the case was built up on what appeared to be convincing grounds and how it had to be abandoned when fresh evidence pointing to the correct aetiology of epidemic dropsy came to light.

Epidemic dropsy clinically is made up of cardiovascular, cutaneous and alimentary manifestations, which superficially appear to be similar to those found in beri-beri. The onset may be sudden or insidious. Premonitory symptoms are nausea, loss of appetite, loose bowels and irregular fever. The diarrhoea is watery and continues to be mild or may become severe. Oedema is confined to legs and feet but may later involve thighs and hands as well; trunk

and face, however, are not affected. The skin is hyperaemic with vascular mottling and petechial rash. The heart is enlarged involving particularly the right side as in beri-beri. Electrocardiogram reveals tachycardia and extrasystoles, shortened P-R interval and an abnormal T-wave suggestive of myocardial degeneration. There are no changes in the nervous system. There is a certain degree of orthochromic normocytic anaemia; blood volume remains unchanged although plasma volume increases; the latter effect may be due partly to plethora and partly to anaemia. Serum calcium is reduced; plasma chloride shows an increase. The disease has a low mortality which may reach 10 per cent in certain epidemics, the cause of death in majority of cases is acute cardiac failure. There is no specific treatment for the disease. Hospitalisation ensures rest and good diet. Medical attention is directed to relief of distressing symptoms and prevention of complications (Chaudhuri and Chakravarti, 1950).

In the early days of the known history of the disease, suggestions regarding an infection being the cause were not wanting, but in the absence of any positive proof the idea came to nothing. Megaw (1923) put forward the suggestion that epidemic dropsy was only a different form of beri-beri. Both diseases were apparently associated with the consumption of rice. Megaw was of the opinion that inefficiently stored rice under humid conditions provided a suitable medium for the growth of fungi and bacteria. These organisms produced a toxin which when consumed for sufficiently long periods brought on attacks of epidemic dropsy or beri-beri. He did not subscribe to the then already proved fact that beri-beri was a deficiency disease. Megaw and Bhattacharji (1924)

reported further studies on 70 cases in Calcutta in confirmation of their fungus-rice hypothesis and Acton and Chopra (1925) lent their weight to the views held by Megaw. They even reported on the properties of the so called toxin, isolated from spoilt rice, the water soluble fraction being responsible for epidemic dropsy and an alcohol soluble factor responsible for beri-beri. Acton and Chopra succeeded in producing oedema in monkeys fed washed rice, the sample of rice being obtained from supplies known to be associated with an outbreak of epidemic dropsy. Other monkeys which received two bananas each per day in addition to washed rice did not develop oedema. It must be said that production of oedema alone is not tantamount to producing epidemic dropsy in monkeys and that protection offered by bananas in continued presence of the hypothetical toxin had no clinical counterpart in human cases. Anderson (1927) carried out a careful survey in an outbreak occurring in a town (population 8,000) in Bengal in September 1926. He failed to find any association between "infected" rice and the disease. Even as late as 1933, Chopra and Bose held on to the view that toxins formed by the activity of *B. vulgaris* on moist rice were responsible for the cardiovascular lesions seen in epidemic dropsy and beri-beri. In certain quarters, however, this hypothesis was received with doubts. Firstly, it was pointed out that clinically the two diseases were different and that there was little doubt that wet beri-beri was caused by a deficiency of vitamin B<sub>1</sub>. In epidemic dropsy there was no peripheral neuritis and although in both the heart was enlarged and oedema was present there were other differences which would distinguish one from the other. In wet beri-beri the oedema can be



generalised as against the oedema of certain selected sites in epidemic dropsy. The skin manifestations and diarrhoea so characteristic of the latter are not known to occur in beri-beri. Further, the symptomatology of epidemic dropsy is suggestive of toxic origin unlike that of beri-beri. Finally the cardiac condition in beri-beri responds dramatically to injections of thiamine hydrochloride, which do not have any effect on patients with epidemic dropsy. These differences between the two diseases came to be grasped only slowly, but fortunately the confusion now no longer exists.

A good deal of confusion was due to the fact that the disease was supposed to be associated with rice. The fact that epidemic dropsy did not occur in all rice eaters even in regions where beri-beri was endemic should have given a lead which was not followed. The curious fact is that the disease almost always occurs in Bengalis. There must, therefore, be one or more constituents of the diet to which Bengalis are partial and other rice eaters are not. Most Bengalis use mustard oil as a cooking fat, hence the interesting observation that in Rangoon, Mauritius, and Fiji the outbreaks were restricted to people who had migrated from Bengal. There were already a few isolated observations directly implicating mustard oil. The prestige and support of stalwarts like Megaw, Acton and Chopra, however, were behind the infected rice hypothesis and no one took heed of the two suggestive observations. Sagayam (1927) in Fiji suspected mustard oil and has recorded that when it was replaced by coconut oil and the mustard by Coleman's mustard no fresh cases occurred. Kamath (loc. cit) while describing the outbreak in Ganjam had referred to adulteration of gingelly oil with an oil.

pressed from 'Odisimari' seed which has now been identified as the seed of *Argemone mexicana*. He knew that local application of argemone oil caused erythema of the skin and local swelling, but did not consider that it had any aetiological role in epidemic dropsy. On the other hand, Sarkar (1926) was quite positive, for he found epidemic dropsy breaking out in families who consumed mustard oil pressed in a country press previously used for pressing argemone oil.

Lal and his colleagues commenced in 1937 an extensive investigation during the course of which careful epidemiological, clinical and other records of various outbreaks between 1937 and 1942 were kept. As a result much light has been thrown on the aetiology of epidemic dropsy. Lal and Roy (1937 a and b) referred again to the alleged role of mustard oil and pursued the matter more vigorously till a large body of evidence was collected implicating contaminated mustard oil as the direct cause of epidemic dropsy. Lal, Ghosal and Roy (1937) examined rice grains from affected and immune localities, but were unsuccessful in isolating *B. vulgaris*. Lal and Roy (1937 c) carried out feeding experiments in human volunteers in a Calcutta Jail in which batches were fed good and 'bad' rice and pure and 'suspected' mustard oil. Whereas volunteers receiving oil of known purity remained healthy throughout the course of the experiment, all the six subjects in the suspected oil group developed, during the course of 10 to 25 days, hyperaemia of skin, oedema of legs, fever and burning sensation over the body. Three out of six showed dilatation of the heart. Examination by more than one experienced clinician confirmed the diagnosis of mild epidemic dropsy. On the appearance of

symptoms, the patients were hospitalized and treated with good diet when complete recovery took place.

Thus the role of mustard oil was convincingly demonstrated. Lal, Ahmad and Roy (1938), however, were of the opinion that allyl isothiocyanate found in mustard oil was not responsible for epidemic dropsy, for its amount in 'suspected' oils was not greater than found in oils known to be harmless. Lal *et al* (1939, 1940) found that the suspected oil gave two reactions which distinguished it from the harmless oil. On shaking with an equal volume of concentrated nitric acid, the acid layer took on a brownish red colour. If the oil acidified with glacial acetic acid was heated on a water bath with a 3 per cent solution of cupric acetate, a precipitate was formed and the aqueous layer changed from brown to green. The former reaction could be quantitatively carried out so as to detect the presence of the toxic substance in any sample of oil. It was also observed that several samples of mustard seed stored for pressing contained varying quantities of seeds of *Argemone mexicana*. The argemone oil itself gave both the above tests, thus lending support to the contention that the presence of argemone oil as an adulterant in mustard oil was responsible for epidemic dropsy in persons consuming the product. The toxicity of the oil seemed to run almost parallel with the strength of the colour obtained with nitric acid. This was later confirmed by Lal and Das Gupta (1941) during an outbreak in a village in north Bengal where the mustard seed was found contaminated with argemone seed and the oil also gave the characteristic test.

The toxicity of argemone oil was tested by Pasricha, Lal and Banerjee (1940) in mice and guinea pigs for whom daily doses of 0.1 and 0.25 c.c. respectively

were found toxic. A dose of 0.54 gm./100 gm. body weight was sufficient to kill a guinea pig. The oil lost its toxicity on being heated for 15 minutes at 240°C. Tissue damage caused by the oil could be demonstrated in intestines, liver and kidneys. The mucous membrane of the stomach and intestines was oedematous, and desquamating in places. Cells showed cloudy swelling. Liver showed portal congestion and thrombosis of tributaries of portal vein, fatty degeneration of parenchymal cells scattered throughout the lobule and necrotic patches near the periphery. Kidney showed acute haemorrhagic glomerulotubular nephritis. Chopra and others (1939) fed five volunteers with mustard oil to which was added argemone oil in concentrations of 2 to 10 per cent and produced oedema in all of them within 15 to 32 days, marked flush in 2 subjects and cardiac symptoms in two. This investigation was followed by the isolation by Lal and his associates (1941) of a crystalline alkaloid as rhombic plates melting at 190°C. It gave the same colour reactions as toxic mustard oil. Feeding human volunteers with mustard oil in which the alkaloid was dissolved did not, however, produce characteristic symptoms even after 23 days of continuous feeding.

Sarkar (1941 a) developed yet a third test for the detection of argemone oil in mustard oil. Equal volumes of the suspected oil and concentrated hydrochloric acid are heated on a boiling water bath for 2 minutes, alcohol is then added, followed by an acid solution of ferric chloride and the mixture is heated and shaken. An orange red precipitate appears indicating the presence of argemone oil. Sarkar (1941 b) found that of 14 oils tested only argemone oil gave this test. He also found that ferric chloride test was



more sensitive than the other two in detecting the presence of small amounts of argemone oil. Later Sarkar (1948) reported the isolation of two alkaloids dihydrosanguinarine and sanguinarine from argemone oil. Of these, sanguinarine was intensely toxic to rats, 2 mg./100 gm. body weight when injected proved lethal; on the other hand, dihydrosanguinarine was not toxic even in doses  $2\frac{1}{2}$  times or more. Sanguinarine administered orally at 1 mg./100 gm. body weight also proved lethal to rats within a week. Sarkar found that sanguinarine inhibits pyruvate oxidation by pigeon brain homogenates and that this inhibition could be prevented by B A L (British Anti-Lewisite). The enzyme system poisoned by sanguinarine could not be reactivated by B A L. The latter exerts its protective action *in vivo* as well. When B A L was injected intramuscularly in rats 15 to 20 minutes earlier than the injection of a lethal dose of sanguinarine, it afforded protection to 85 per cent of the animals who would otherwise have died. These observations are indeed very interesting and places the role of argemone oil in the aetiology of epidemic dropsy on a surer footing than before. They also explain the failure of Lal *et al* to produce toxic symptoms in rats by administration of the crystalline substance isolated by them for it is probable that it was the non-toxic (or comparatively so) dihydrosanguinarine. The latter forms 87 per cent of the total alkaloids present in argemone oil, the toxic action is probably due to the small proportion of sanguinarine that is present. It is not beyond the reach of probability that dihydrosanguinarine could be oxidised to sanguinarine in the body and then prove toxic. More evidence on the subject would be welcome.



People often wonder how mustard oil gets contaminated with argemone oil. It has been observed that not infrequently mustard seed intended for pressing is found mixed with *Argemone mexicana* seeds. This mixture can hardly be considered fortuitous for some very valid reasons. *Argemone mexicana* is a weed which grows wild in jungles and even on bunds of cultivated lands. It is not necessarily partial to fields sown with mustard. Even assuming that it was, the plant flowers and bears fruit much later in the season than the time when the mustard crop is harvested. One cannot escape the conclusion that certain unsocial elements make it their business to collect argemone seed and mix it with mustard seed. The only thing that is common between black mustard and the former is that both are black in colour and hence an unwary customer may not easily detect adulteration of the seed. The practice of adulteration is nefarious and dangerous to public health. The ferric chloride test referred to above has been utilized by the State Laboratories for the detection of argemone oil in mustard oil. A tolerance limit of 0.5 per cent has been set, but even this should not be permitted in the interest of public health. Apart from exemplary punishment meted out to the miscreants, recent attempts to eradicate the weed and thus remove the source of trouble are likely to prove of lasting benefit to the community.

### LATHYRISM

Among the diseases associated with the consumption of certain foodstuffs lathyrism stands out as one of great importance in India from the health and economic standpoints. There is extensive cultivation in India of *Lathyrus sativus* known by several vernacular

names such as *teora*, *khesari*, *lakh* or *lang dahl*. The Central Provinces (now known as Madhya Pradesh), parts of Central India now included in the states of Madhya Bharat and Vindhya Pradesh, eastern parts of the United Provinces, Bihar and to a certain extent North-west Frontier Province of Pakistan are the regions where *L. sativus* is cultivated. The plant belongs to the order Leguminosae and gives pods containing black seeds of the size of a pea. The seed coat is black and on splitting yields a pulse which looks not unlike the Bengal gram pulse. It can be cooked as pulses usually are cooked in India or its flour can be used either alone or in mixture with wheat for making thick unleavened bread. The crop has obvious advantages from the cultivator's point of view. It is comparatively hardy, can be grown in fields or on bunds surrounding the rice field. It withstands drought to a considerable extent and thrives under conditions where a crop like wheat would certainly fail. It is these characteristics that make for its abundant growth and its use as a human food for poor people at all times, so much so that during periods of famine and scarcity, it forms a staple article of the diet in certain regions. The chemical composition of the pulse analysed at the Nutrition Research Laboratories is given in Table XXVII.

In its nutritive value the *L. sativus* protein does not markedly differ from protein of some other pulses although it is markedly inferior to the proteins of Bengal gram and red gram. In spite of these considerations and in view of its relative abundance and low price, lathyrus pulse has enjoyed considerable popularity in certain regions of India.

TABLE XXVII  
COMPOSITION OF THE DECORTICATED SEED OF  
LATHYRUS SATIVUS

Contents		Per 100 Grams.
Moisture	...	10.0 gm.
Protein	...	28.2 „
Ether Extractives	...	0.6 „
Mineral matter	...	3.0 „
Carbohydrate (by difference)	...	58.2 „
Calcium	...	0.11 „
Phosphorus	...	0.50 „
Iron	...	5.6 mg.
Carotene	...	200 $\mu$ g.
Thiamine	...	125 „
Riboflavin	...	414 „
Nicotinic Acid	...	1.2 mg.

The biological value of *L. sativus* protein is rather low. The following figures are taken from Basu, Nath and Mukherjee (1937) who used albino rats for the investigation.

		Level of intake	
		10%	15%
Digestibility co-efficient	...	90	90
Biological value (Balance Sheet method)...		50	44
Do. (Growth method)	...	—	0.6

The association of the consumption of lathyrus pulse with the disease now known as lathyrism was first brought to light in India by General Sleeman in 1844. His graphic description given over a century ago is worth quoting in full:

“In 1829 the wheat and other spring crops in Saugor and surrounding villages were destroyed by hailstorms and rains and in 1831 they were destroyed by blight. During these three years the “teori” or what in other parts of India is called “kesari” (*Lathyrus sativus*), a kind of wild vetch which, though not sown itself, is left carelessly to grow among the wheat and other grains and given in the green and dry state to cattle, remained uninjured and thrived with great luxuriance. In 1831 they reaped a rich crop of it from the blighted wheat fields and subsisted upon its grain during that and the following years, giving the stalks and leaves only to their cattle. In 1833 the sad effects of this food began to manifest themselves. The younger part of the population of this and the surrounding villages, from the age of thirty downwards, began to be deprived of the use of their limbs below the waist by paralytic strokes, in all cases sudden, but in some cases more severe than others. About half the youth of this village of both sexes became affected during the year 1833-34 and many of them have lost the use of their lower limbs entirely and are unable to move. The youth of the surrounding villages in which the “teori” from the same causes formed the chief article of diet during the years 1831-32 have suffered to an equal degree. Since the year 1834 no new case has occurred but no person once attacked had been found to recover the use of limbs affected and my tent was surrounded by great numbers of the youth, in different stages of the disease,

imploing my advice and assistance under the dreadful visitation. Some of them were very fine young men of good caste and respectable families and all stated that their pains and infirmities were confined entirely to the part below the waist. They described the attack as coming on suddenly, often while the person was asleep, and without any warning symptoms whatever and stated that a greater portion of young men were attacked than of the young women. It is the prevailing opinion of the natives throughout the country that both horses and bullocks, which have been much fed upon "teori" are liable to lose the use of their limbs, but if the poisonous quantities abound more in grain than the stalk or leaves, a man who eats nothing but the grain must be more liable to suffer from the use of the food than beasts, which eat it merely as they eat grass or hay."

Records of outbreaks of lathyrism between the time when Sleeman's description appeared and the first decade of the present century are not readily available unless one searches the archives of some provincial health departments. From 1904 onwards, however, one finds frequent mention of the disease having occurred in sporadic or epidemic outbreaks in several provinces. Buchanan (1904), Acton (1922) and McCombie Young (1927) report its occurrence in the Central Provinces; McCarrison (1926) and Mackenzie (1927) have described it in Gilgit Agency in Kashmir, Stott (1930) in Uttar Pradesh (United Provinces) and Shourie (1945) in Bhopal State in Central India. In the year 1946-1947 the Central Provinces reported its occurrence in an epidemic form involving about 7,000 individuals. Although detailed information from Bihar is not available, lathyrism is undoubtedly endemic in some districts in the province.



Lal (1949) describes three outbreaks in the districts of Patna, Monghyr and Darbhanga in Bihar province.

Acton's description of the disease and the discussion of its aetiology is the best that is available from recent observations. He confirmed Buchanan's estimate that in certain districts the incidence may be as high as 6 per cent of the population. Acton examined 204 cases between the ages of 3 to 60 years. Of these 170 were between 15 and 30 years of age, 181 were males and 23 females. The higher incidence of the disease among males and in young adults has been confirmed by later observations as well. Acton found that in Rewa and Sutna towns, the largest number of cases appeared in July. This he ascribes to the larger consumption of lathyrus during the three or four preceding months than in the rest of the year.

**Clinical Features:** Acton describes four stages of the disease. The first stage is characterised by weakness of the lower limbs with spasticity of several muscles so that movements at the ankle and knee joints are restricted and painful. The knee and ankle joints both are held in a partially flexed position so that when the patient walks he assumes a characteristic posture and gait. There is no stamping or swinging round of the foot while walking.

In the second stage, flexion of knee is more marked, there is a certain amount of inversion of the foot with tendency to walk on toes. The spasm of adductor muscles causes one foot to be brought in front of the other during progression which is with the help of a long stick.

In the third stage the symptoms described above become more marked and the patient can only walk with the help of crutches or sticks (Fig. XXV).

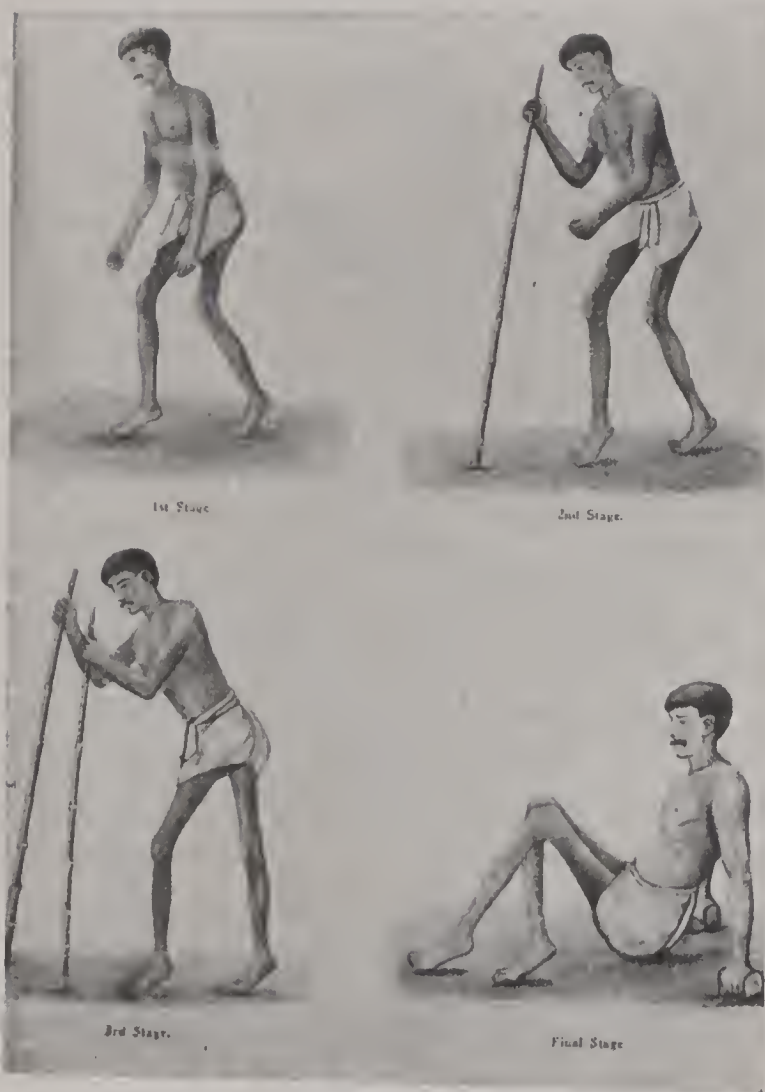


Figure XXV—Four stages of lathyrism: See description in the text.  
(By courtesy of the Indian Medical Gazette).



In the fourth stage the knees become completely flexed and erect posture and walk become impossible.

There is atrophy of the thigh and leg muscles, knee jerks are brisk and plantar reflex is extensor.

In the upper extremities and the rest of the body there is no motor disturbance and function is normal. At no time is there any sensory disturbance such as calf tenderness, or numbness, tingling, pain, etc., in the affected limbs or elsewhere. Sphincters are usually not involved and mental condition remains normal throughout. The disease gives an impression of being due to the involvement of pyramidal tract probably in the proximal lumbar region.

The onset in most cases is believed to be sudden, for instance, the attack may come on while working when the person may suddenly feel the loss of power in his legs and fall down. In other cases patients may find themselves unable to get up after a night's sleep before which they had been perfectly normal. It must be said, however, that not enough is known of the latent period and mode of onset for lack of careful observation. The progress of the disease as mentioned before may ultimately cripple the subject. The patients, whether they are in the early stages or late, are not known to recover the normal function of the lower extremities at any time during their life.

Lathyrism is not peculiar to India alone. Wherever *L. sativus* is used as human food, its outbreaks have been reported—e.g. in France, Germany, Italy, Algiers in North Africa, Russia and more recently in Spain. From the last mentioned country several reports are available but none of them throw additional light on the problem. Diaz *et al* (1942) have referred to the paucity of information on morbid anatomy of the disease. The reports of autopsies mentioned by

them are not available with the exception of one by Filimonov who kept a lathyrism patient in his clinic for 30 years before he died of acute leukaemia. In the absence of the original publication, the interpretation of Diaz that lesions of the spinal cord in the third lumbar segment account for the clinical signs of the involvement of the pyramidal tract has to be taken as probably correct. Clearly further information on this aspect is essential for any progress to be made in the understanding of the disease.

There is practically no information from Indian sources on the pathology of the disease, for there has been no post mortem examination even of a single case. The reason for this probably is that the afflicted patients being incapable of earning a livelihood migrate and wander from place to place as beggars. They ultimately succumb to some intercurrent infection. Their death is not due to lathyrism and as yet no attempt has been made to keep track of such cases and to study the anatomical lesions responsible for the disability, although on theoretical grounds the pyramidal tract has been considered to be involved.

The attempts to produce the disease experimentally in laboratory animals have not at all been successful. McCarrison (1928) fed to rats *L. sativus* obtained from areas where lathyrism cases had occurred. Wheat and lathyrus were fed in different proportions but even on 100 per cent lathyrus diet fed for 200 days no symptoms could be produced in rats. This experiment was later repeated by McCarrison and Krishnan (1934) with a sample of pure strain of *L. sativus* with the same result. In 1946, Patwardhan once again used a market sample of the lathyrus gram and found that rats were not affected. There was a popular belief that lathyrism may be caused by



consumption of seeds of *Vicia sativa* which are often found admixed with lathyrus crop. Anderson, Howard and Simonsen (1924) conducted experiments with pure strains of *L. sativus* and *V. sativa*. The latter when fed to monkeys produced hyper-irritability of muscles and muscular clonus with symptoms like tetany. In ducks on *V. sativa* diet, symptoms resembling avian polyneuritis were produced. The feeding of *L. sativus* on the other hand was harmless. The condition described in the monkey does not appear to resemble lathyrism as seen in human beings. Acton and Chopra (1922) claimed to have isolated an active crystalline compound which in guinea pigs produced a transient spastic paralysis from which the animals recovered. The authors suggest that the same principle may be active in human lathyrism. Stott (1930) fed pure strain seeds of *L. sativus* and *Vicia sativa* to ponies for  $4\frac{1}{2}$  months. During that time symptoms of lathyrism did not develop. The experiment had to be concluded as the stock of the two grains was exhausted. Bhagvat (1946) found that if guinea pigs were fed a diet containing lathyrus pulse to the extent of 50 per cent or over, their growth was retarded and the guinea pigs became dull and weak with disinclination to move; they also developed trophic ulcers on hind legs and died after a few weeks; the cause of death could not be determined. Even allowing for species difference in response, the above symptoms cannot be considered as in any way resembling lathyrism. It must be admitted that thus far animal experiments in India have thrown no fresh light on the subject.

In Spain, Diaz and Vivanco (1942) fed rats, dogs and monkeys on diets containing *L. sativus* obtained from regions where outbreaks of lathyrism had occurred. They could not produce symptoms resembling

lathyrism in any of the three animal species studied by them.

In America, Geiger, Steenbock and Parsons (1933) showed that rats fed a diet containing over 50 per cent of *L. odoratus* (sweet pea) developed certain toxic symptoms. These consisted of lowered growth rate, marked spinal curvature, deformity of sternum, deformity in long bones with exostoses, shambling gait and in a few animals paralysis of hind legs. In young animals vitamin supplements did not affect the toxicity of the diet. Lewis *et al* (1948) confirmed the findings of Geiger *et al* and extended them. These authors found that *L. sativus*, *L. cicera* and *L. aphaca* did not produce any toxic symptoms in rats whereas *L. hirsutus*, *L. sphericus*, *L. tingitannus* and *L. sylvestris Wagneri*, were toxic, the last named being the most toxic. They also observed that extra casein in the ration did not alter the toxicity. The toxic principle was extractable with cold water or 30 per cent alcohol, but was insoluble in ether or absolute alcohol. The legume meal from which the active principle had been removed was not toxic, but could be rendered so by addition of the concentrated extract. Lewis and Schulert (1949) and Lee (1950) found two other species of lathyrus, viz., *L. latifolius* and *L. pusillus*, to exert similar toxic effects in rats. Lee observes that the toxin is heat stable and is not an enzyme. Feeding with vitamin E gave a certain protection against paralysis.

The results of Geiger *et al* and Lewis *et al* thus throw light on the failure of McCarrison to produce toxic symptoms in rats by feeding with *L. sativus*. Recent experiments at Coonoor with monkeys and dogs point to the conclusion that *L. sativus* may be nontoxic to other species as well. It must be admitted,

however, that clearcut evidence can only be obtained when other species are fed pure strains of different species of lathyrus. A re-examination of certain aspects of the evidence available from studies on lathyrism outbreaks is suggestive of the fact that possibly in human beings too *L. sativus* is nontoxic and that lathyrism is the result of contamination of *L. sativus* crops with one or more toxic species of lathyrus. A few enquiries made to elicit information on the occurrence of such contamination in the Indian lathyrus crops have given encouraging results. It has been reported that in the United Provinces where *L. sativus* is grown as a crop, *L. sphericus* has also been found to occur. Similarly in Bihar where the examination of a few samples of khesari gram was carried out in the Directorate of Agriculture at our request it was found that *V. sativa* (1.33 per cent) *V. hirsuta* (0.61 per cent), *L. aphaca* (14.08 per cent) and *L. sphericus* (2.28 per cent) were present as contaminants. Of the above, the grains of vicia can be separated by mechanical cleaning. Besides, experiments referred to earlier have shown that although vicia contains poisonous alkaloids, their action does not produce symptoms in animals reminiscent of lathyrism. Of the remaining two contaminants, *L. sphericus* was present in small quantities. The question now to be decided is whether its presence in such quantities would give rise to a condition like lathyrism. The experiments of Geiger *et al*, Lewis *et al* and Lee are not strictly comparable with the events that lead to human lathyrism, although they were effective in showing up the harmlessness of *L. sativus* and toxicity of some other species of lathyrus. It will be necessary to carry out experiments with other species of animals with *L. sativus* alone and mixed in

different proportions with a toxic species like *L. sphericus*. The reason for selecting the latter as the representative of toxic varieties is the fact that apart from *L. odoratus* which is a garden plant, it is the only one of the toxic species which has been reported to occur in India. Such an investigation is being planned in India and is to be undertaken in conjunction with a field and clinical study of the disease in the endemic regions.

The Governments of the regions concerned have at various times taken halting steps to prevent the disease. The ruling prince of a state in Central India, once forbade paying the agricultural labourer in kind which in that region was entirely in the form of lathyrus grain. Four years back the Government of Central Provinces ordered that lathyrus shall not be sold unless in a mixture with other food grains in which its proportion shall not exceed 33 per cent. The Government of Madras have completely banned the import and sale of lathyrus within Madras Province.

These measures are at best of temporary value. They do not solve the problem, for lathyrus crops are still being extensively cultivated and it is extremely difficult to stop poor people from having recourse to this cheap variety of grain although they know that its consumption is possibly fraught with danger. Even these people have come to associate the disease with the consumption of grains cultivated only in certain areas. There might be some truth in it, for Acton has mentioned that in Rewa and Sutna (Central India) the locally grown variety was probably nontoxic, the disease making its appearance two to three months after the people had consumed the grain imported from Bhagalpur (Bihar). This observation also lends support

to the "contamination hypothesis" of the aetiology of human lathyrism mentioned above.

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## CHAPTER XXI

### FLUOROSIS

IN April 1936 the health inspectors (the junior-most officials in the hierarchy of the provincial public health directorate) of Podili and Darisi taluks of Nellore District in Madras Province brought to the notice of their District Health Officer, the occurrence among the inhabitants of a disease characterised by pains in the cervical and lumbosacral regions of the spinal column and pain and stiffness in joints of both extremities. This preliminary report brought to light a disease later identified as fluorosis, and led for a time to intensive investigation into its incidence, clinical features and aetiology. A preliminary account of the conditions in Nellore District was given by Shortt, Pandit and Raghavachari (1937) which was soon followed by detailed observations by Shortt, McRobert, Barnard and Nayar (1937). The following description is taken from the account given by these authors.

The earliest evidence of fluorosis is found in mottling of teeth which appeared to be very common in children born and brought up in the endemic area. Mottling is usually seen in the permanent teeth, occasionally, however, deciduous teeth may also be involved in regions where the fluoride content of water is high. The enamel loses its lustre and becomes chalky white and at places bands of chocolate brown pigmentation may be found. There may be pitting as well. Mottling is observed best in the central incisors of the upper jaw, but other teeth in the upper or both the jaws may be involved. The children do





Figure XXVI—An advanced case of fluorosis showing spinal deformity and rigidity.

*(By courtesy of the Indian Journal of Medical Research).*



not show any other evidence of fluorosis and appear, except for mottled teeth, similar in all respects to children belonging to the nonendemic areas.

The next stage in the disease among those who continue to live in the region makes its appearance between 25 to 30 years of age. First to appear is the recurrent tingling sensation in the limbs or over the whole body. This is followed by pain and stiffness in the lumbar regions of the spine, but may also extend to the dorsal and cervical regions. The stiffness increases till the spine becomes almost a rigid column. If the patient has to look sideways, he may have to turn the head with the trunk in the desired direction. Bending of the back becomes impossible. The other joints particularly of the lower limbs are also affected, and the thorax becomes more or less fixed, leading to abdominal breathing. The patients assume a characteristic posture and gait. It takes nearly 5 to 10 years before the full picture is developed. This is accompanied by progressive loss of appetite and general wasting, loss of sphincter control and impotence. The patient finally becomes so immobile that he becomes bed-ridden and succumbs to some intercurrent infection (Fig. XXVI).

The clinical findings in the examination of cardiovascular, respiratory, alimentary and nervous systems did not reveal anything abnormal. Major changes were found in bones and joints. There was synostosis of the entire vertebral column, starting with the lumbar region. Kyphosis or scoliosis was present in some. In most, the costochondral junctions were ossified. There were bony outgrowths along the tendinous insertions of muscles in the long bones. Osteophytic growths were also seen at the borders of the ribs; the interosseous membranes between the

long bones were partly or wholly calcified. X-ray examination also showed increased density of bone with exaggeration of the trabecular pattern and decrease in the medullary cavity. The movements of joints, especially hip, knee, shoulder, elbow and ankle joints, were limited and painful. Certain biochemical investigations were done in ten subjects. Blood sugar, cholesterol, and serum magnesium were within normal limits. But the average values for serum calcium and inorganic phosphorus were slightly above the upper normal limit, so was the serum alkaline phosphatase. Kidney function appeared to be impaired in most. In three cases the fluoride content was 1.82 mg. per 100 cc. blood and in the urine of six cases the average fluoride content was 1.99 mg. per 100 cc.

The above investigation was followed up by the examination of water supplies from all over the Madras Province. About 1,747 water samples from 24 districts were analysed by Raghavachari and Venkataramanan (1940) for their fluorine content; the results are shown in Table XXVIII.

Thus the water of five Ceded Districts and of Nellore especially seemed to be rich in fluoride. The highest fluoride content was found in Darisi, Podili and Kanigiri taluks of Nellore District, i.e., the regions where bone fluorosis incidence is at its highest. The map of the Madras Province given in Fig. XXVII clearly illustrates the relation between the fluoride content of water and incidence of fluorosis. Even within the endemic areas there are wells with water containing no fluoride or fluoride only in traces. One finds people migrating from one settlement or village to another site in search of fluoride free water, for they have come to associate the disease with water

TABLE XXVIII—FLUORIDE CONTENT OF WATERS  
IN MADRAS PROVINCE

Region	Total Number of Samples	No. of samples with Fluoride Content of			
		Nil or Trace	Under 1 p.p.m.	1 to 3 p.p.m.	Over 3 p.p.m.
Anantapur District...	365	22	58	269	16
Bellary           ,,   ...	50	15	23	12	...
Cuddapah       ,,   ...	68	15	19	32	2
Guntur           ,,   ...	36	8	6	18	4
Kurnool          ,,   ...	31	6	9	13	3
Nellore          ,,   ...	611	98	106	349	58
Total for Madras Province (including other districts not mentioned in the table)           ...	1747	522	339	795	91

supply even without the scientific background of knowledge. Pandit, Raghavachari and Subba Rao (1940) made a further study of the causative factors in endemic fluorosis. They found that the incidence of mottled teeth in Anantapur and Nellore Districts varied from 50 to 90 per cent. In Cuddapah it was 60 per cent and in a Bellary Famine Camp the incidence was 70 per cent. In Nellore the incidence of mottled teeth could be correlated with the fluoride content of water. Bone lesions also varied accordingly, in one particular area within Nellore District, the authors found 74.4 per cent of adult population of 1,192 suffering from bone fluorosis. The authors carried out a diet and nutrition survey among the

population. The diets were deficient in protective foodstuffs and hence there were deficiencies of several nutrients particularly of vitamins A and C. The authors were of the opinion, however, that ascorbic acid deficiency existed in greater measure throughout

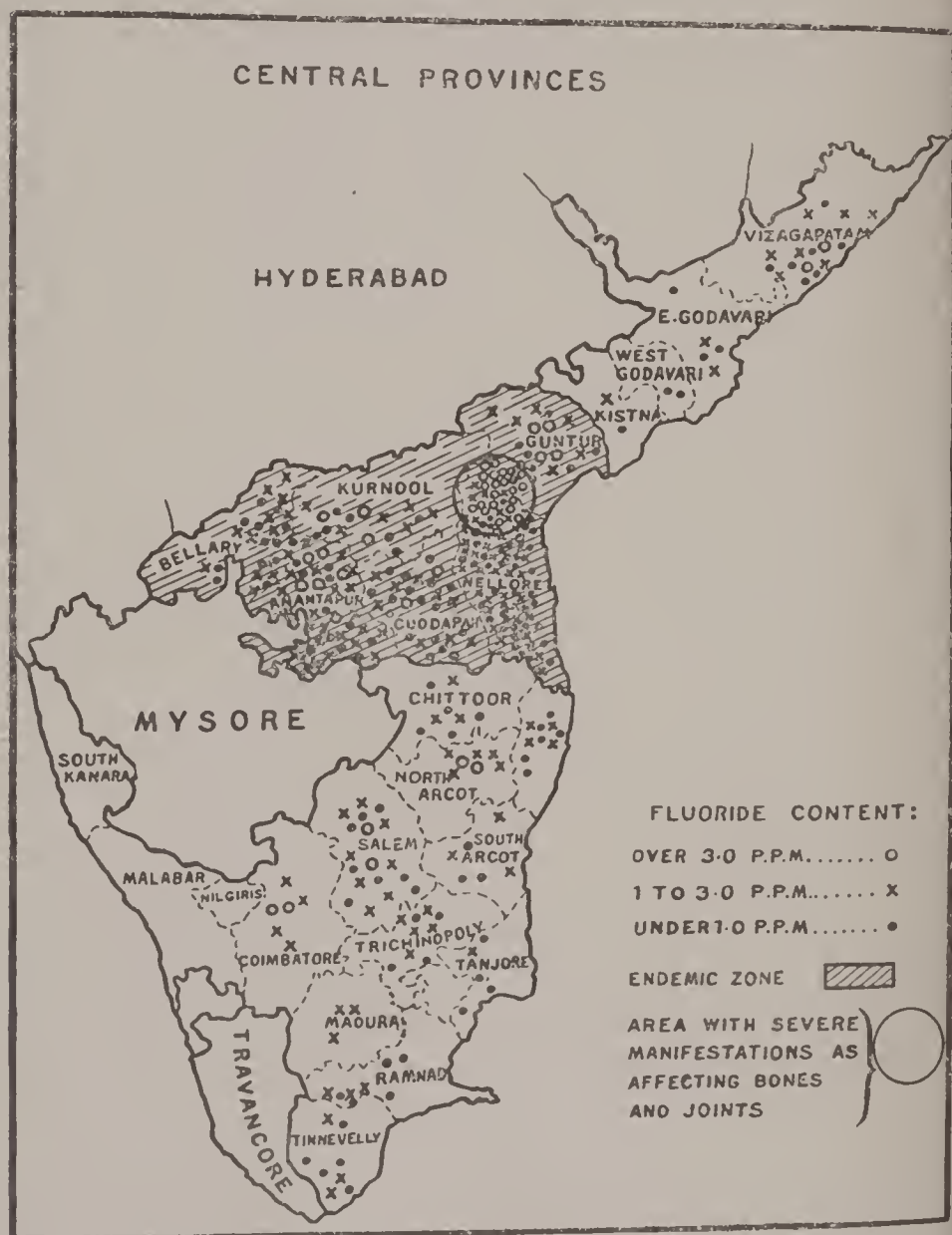


Figure XXVII—Fluoride content of waters in Madras State the endemic zone of fluorosis.

(By Courtesy of Indian Journal of Medical Research.)

the year. This could not be substantiated by clinical findings although other signs of malnutrition such as phrynoderma, rickets, angular stomatitis, xerosis, etc., were present to a marked degree. They also observed that among the economically higher groups of people bone fluorosis was rarer as compared with its occurrence among poorer classes of the endemic region. This difference was ascribed to a better dietary of the former group.

The highest amount of fluoride found in water in the endemic region has not exceeded 6 p.p.m. In view of this it is indeed surprising that lesions of such severity as described above develop in the local population. This is not in keeping with the experience in the United States of America where fluorosis of such severity has not been reported in spite of the fact that water supplies containing as high as 12 p.p.m. are known to occur in certain parts of the country. This is probably due to the fact that effective methods of freeing water of fluoride may have been undertaken to protect the community. Bone lesions of fluorosis have only been reported among cryolite workers in Scandinavia by Roholm (1937) whose description of the disease is well known.

Fluorosis in other parts of India also has been reported. In Raichur, Mahboobnagar and Nalgonda Districts of Hyderabad State bordering on the endemic areas of Madras Province, mottling of teeth in children has been observed, the fluoride content of water varied from 1 p.p.m. to 5 p.p.m. (Daver 1945). Wilson (1939) and Day (1940) observed mottling of teeth in Punjab and Pillai (1942) in Travancore, Khan (1945) has described the occurrence of bone fluorosis in human beings and cattle in Punjab.



A certain amount of work on the role of diet on the progress of fluorosis has been carried out in animals. Pandit and Rao (1940) produced chronic fluorine intoxication in monkeys. They found that in monkeys kept on ascorbic acid deficient diets the lesions were more severe, a finding which supported the observations of Pandit *et al* (loc. cit) that in the population affected most by fluorosis a dietary deficiency of vitamin C existed. Ranganathan (1944) found that addition of calcium salts had an ameliorative effect on fluorosis induced in rats. Pillai, Rajagopalan and De (1944) observed that whole milk powder added to a fluorosis promoting diet afforded protection to rats. It must be admitted, however, that the above work has not yet carried our knowledge much further on the way to the understanding of the role of one or more dietary deficiencies in the aetiology of fluorosis.

The condition of the people in the endemic areas todate remains unchanged. The old cases are progressing slowly to the inevitable end and new cases are appearing on the scene. No preventive measures are known to have been taken by the Government concerned and one does not know when they will be taken. This is indeed a sad commentary on our public health administration.

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## CHAPTER XXII

### NUTRITION IN PUBLIC HEALTH PRACTICE

THE CONTRIBUTION made by the State Public Health Services in the fight against malnutrition has been extremely meagre, so meagre in fact that no remarkable achievement can be cited on the credit side of the ledger. This is indeed astonishing in view of the fact that laboratory researches and investigations in the field have demonstrated the crying need for some action. The information given in detail in the preceding pages must convince anyone that whereas nutrition research in India has progressed steadily and satisfactorily, its application in public health practice for the benefit of the people has lagged behind considerably. It may be worthwhile examining the causes of the inertia, apathy or inability—one or all of them—of the State to undertake measures to improve the nutrition of the masses.

It was in 1864 that the foundations of a public health service in India were laid by the appointment of Sanitary Commissioners for the three Indian provinces of Bengal, Bombay and Madras. Prof. Gangulee (1938) in his book entitled *Health and Nutrition in India* has described in brief the development of Public Health Service in the provinces and at the centre. The Report of the Health Survey and Development Committee, Government of India (1946) has also traced the history of public health administration in India. It is not our purpose to go over the same ground again. It must be mentioned, however, that organised public health activity in India has been halting in its progress till 1919 and its efforts on the

health of the community not very impressive. There were some difficulties inherent in the situation. The public health organization in the country was primitive till 1904. Further, the administration of public health necessitated legal sanction and it is possible that in the early days the government policy of not interfering unduly with the habits and customs of the Indian people and their mode of life may have set the pace at which reforms intended to improve environmental hygiene could be put through. The epidemic of plague which swept India in the closing years of the last century and the Report of the Plague Commission published in 1904 awoke the people and stirred the Government to strengthen the then existing public health administration. When the Government of India Act of 1919 came into force a large measure of autonomy in internal health administration was granted to the provinces. This fact did contribute to the expansion of public health services and the efforts of the provincial administration did to a certain extent improve the environmental hygiene and sanitation mainly in urban areas. The villages, however, benefitted but little. Several factors may have contributed to this state of affairs. The enormous population, extreme poverty of the masses coupled with wide-spread illiteracy and ignorance born of it and a complete lack of sense of social hygiene were tremendous obstacles to overcome. Added to these was the ever present financial stringency experienced by provincial administrations. Hence no wonder that Public Health Department was one of those which could not undertake any ambitious programmes for the improvement of health. Finally the artificial division of the country—administratively into British India and Princely India—left gaps too large to permit any

uniform policy being considered for the country as a whole.

The State Public Health Services have been primarily concerned with the improvement of hygiene and sanitation and with the fight against infectious and communicable diseases. Diseases like small-pox, plague, cholera, malaria, kala azar, etc. have their endemic foci within the country and given favourable conditions they not infrequently break out in epidemics calling for the resources of the State for meeting the contingency. It can be said without exaggeration that prevention of epidemics of such diseases has been the major function of the Public Health Departments. They had not the resources to eradicate these diseases or effectively control them. Greater is the pity since the more advanced countries have shown what can be achieved in this field.

Till recently nutrition as a part of the state public health responsibility was not in the picture. It is true that the role of nutrition in the fight against disease was scientifically demonstrated only in the twentieth century. With it came the knowledge about the various early and late manifestations of nutritional disorders and a realisation that much of morbidity and mortality in countries such as India would be due basically to malnutrition. It was the duty of the state to organise collection of information on the state of nutrition of the population, particularly the vulnerable groups in it, and to devise ways and means of improving the standard of nutrition wherever necessary. During the last twenty years or more, several countries in Europe and America took advantage of scientific knowledge available and took necessary steps in this direction. Even during times of extreme stress such as the last war, provision was made in these countries



to feed their peoples, as far as possible, on sound nutritional lines. To give only one example, people in the United Kingdom were scientifically fed and the nutrition of vulnerable groups carefully looked after with the result that after six years of a life and death struggle the country emerged in a condition of health in some respects even better than before the war.

The researches of McCarrison and the observations of the Royal Commission on Agriculture (1926) on their significance made the Government of India nutrition-conscious for the first time. A little later the Far Eastern Conference on Rural Hygiene which met at Bandoeng, Java, managed to impress upon the participating governments the significance of malnutrition as a public health problem and the importance of taking steps to tackle it effectively. In 1937, the Provincial Governments in India took the first step in this direction, i.e., to depute personnel for training at the Nutrition Research Laboratories, Coonoor, where a course on theoretical and practical nutrition was held for the first time in the summer of 1937 and then annually thereafter. It was the intention of these governments to appoint the trained personnel as nutrition officers in charge of public health nutrition work within the province. However, it is sad to have to record that a very small proportion of the persons thus trained is today actually employed in public health nutrition work.

The organisation and activities of nutrition sections in state governments are given in Table XXIX.

It will be clear from the Table that it took eleven years to have nutrition sections opened in 13 provinces and states, and that before World War II only three governments had any kind of nutrition organisations. Subsequent to 26 January 1950 the

TABLE XXIX—NUTRITION ORGANISATION IN THE INDIAN STATES

State	Nutrition Section organised in the year	Organisation directed by	Type of Activity	Remarks
1. Assam	....	1947	Nutrition Officer	The section retrenched within a year of formation.
2. Bengal	....	1937	"	The section was discontinued within a few months after its inauguration.
(a) West Bengal	....	1950		Post-partition province.
3. Bihar	....	1938	"	
4. United Provinces	....	1948	"	
5. Delhi	....	—	—	
6. Punjab	....	1938	Nutrition Officer	After partition, nutrition section established in 1948.
7. Orissa	....	—	—	
8. Central Provinces	....	1945	Nutrition Officer	
9. Hyderabad	....	1939	"	
10. Bombay	....	1944	"	
11. Mysore	....	—	—	
12. Madras	....	1944	Nutrition Officer	
13. Travancore	....	1941	Superintendent, Public Health Laboratory.	
			Field Work, Advisory	
			Field Work, Advisory, Laboratory investigations.	
			Field Work, Advisory	
			Field Work, Advisory	
			Field Work, Advisory	
			Field Work, Advisory and Laboratory investigations	
			Field Work, Advisory	
			Field Work, Advisory, Laboratory investigations	

Republic of India has been divided into states of three categories. Part A States are the regions which were formerly known as provinces in which have been incorporated some princely states which chose to merge with the neighbouring provinces. Part B States are the old large princely states singly or their unions wherever they were formed. In area and income they do not compare unfavourably with Part A States. Part C States are centrally administered areas. These are small and distributed throughout the country. The number of existing nutrition organisations in the whole country is shown below:

	Part A	Part B	Part C
Total number of States ....	9	9	9
Number of nutrition organisations ....	7	2	nil

Most of these organisations follow one pattern. The nutrition section is a part of the public health directorate and is under the charge of a nutrition officer, assisted by field technician staff and some administrative staff. Only in Bihar was the section provided with laboratory facilities right from its commencement. Recently, Bombay has organised its nutrition section on a more liberal scale adequately staffed and provided with laboratory and hospital facilities. Madras State has also reorganised its Nutrition Bureau with a view to make it a more efficient organisation than it was before. The Travancore organisation was not badly organised. But less said about other provincial nutrition organisations the better. A very brief account of the work done by each of the above mentioned nutrition sections will

now be given before making further observations on the subject.

**Assam:** One diet and nutrition survey had been carried out among tea garden labourers before 1947. A few more diet surveys have been carried out since then. Mild chronic malnutrition was found prevalent among the poorer people.

**Bengal:** The Nutrition Section was short lived and hence no work was done. The Provincial Department of Health is, however, assisted by a Nutrition Committee, consisting of official and non-official nutrition experts. There is no indication that the establishment of this Committee had any effect on the inertia of the Government to undertake work in public health nutrition. On one occasion, however, the advice of the Committee was helpful to the Government. It was in connection with the problem of dealing with the starved and destitute population flowing into Calcutta from the rest of the province during the tragic days of Bengal famine in 1943.

**Bihar:** During the twelve years of its existence the nutrition section carried out diet surveys of over 5,200 families in urban, rural and industrial areas. Some repeat surveys were also done to evaluate the effect of food shortage and rising prices on the food habits of the population. In the earlier surveys it was revealed that with an increase in income the consumption of protective foods showed an increase. Over 42,000 children were examined for the evaluation of nutritional status. The results have been referred to in another chapter. In the laboratory, the composition of over 300 foods was determined; nutritive value of certain animal and vegetable proteins was evaluated. Other activities of the section

were directed to nutritional education and propaganda.

**Uttar Pradesh (United Provinces):** Nutrition surveys in school children in four districts have been carried out.

**Punjab:** Diet and Nutrition surveys were carried out in several districts of the province. Recently nutrition surveys have been conducted in relief camps for displaced persons (refugees from Pakistan). An attempt was made during the early years of World War II to provide iodized salt to the inhabitants of Kangra Valley where goitre is endemic. The results of this experiment are, however, not available.

**Orissa:** Although this province has not yet organised a separate nutrition section, the public health department did, however, carry out between 1941-1945 diet and nutrition surveys and repeat surveys in some typical localities.

**Madhya Pradesh (Central Provinces and Berar):** A few diet and nutrition surveys were carried out in urban and rural areas. Some of these were repeated. Supplementary foodstuffs and multi-vitamin tablets were distributed. Other activities included nutritional education and propaganda.

**Hyderabad:** Extensive diet surveys in rural and urban areas have been carried out. Data are available for over 7,500 families. 50,000 school children have been examined for their nutritional status. The section has also attempted to propagate knowledge in nutrition.

**Bombay:** It is interesting to note that a nutrition section in the province was established following the introduction of cereal rationing in 1943. The section has gradually developed and today is one of the best equipped and staffed in the country. Dietaries



of residential institutions controlled by Government have been studied and revised. The section also watches over the nutrition of relief camps for displaced persons. Facilities have recently been extended for the study of nutritional diseases. In the laboratory, investigations on the nutritive value of Indian foodstuffs and relation between poor diet and liver disease are being conducted.

**Madras:** The Nutrition Bureau has been till recently composed of only two regional field investigation units and a co-ordinating officer at the headquarters. The work has been entirely concerned with diet and nutrition surveys. Such surveys have been carried out in almost all the districts of the province and within recent years have provided authoritative information on the dietary habits and the state of nutrition over the whole province. In fact, Madras is the only State which possesses adequate information on the diets of its population. The Bureau is also engaged in education and propaganda work as a part of the campaign for the improvement of nutrition of the masses. Recently the Nutrition Bureau has been reorganised with a view to enable it to play an effective role in the attempts to improve the nutrition of the people.

**Travancore:** Diet and nutrition surveys in families and institutions have been carried out. The nutritional state of over 21,000 children has been determined.

The only activity of importance in which the various nutrition sections had been engaged was, as already mentioned, the diet and nutrition surveys. No doubt they collected valuable information on dietary habits and the state of nutrition of the population, particularly of school children. One can

legitimately ask, however, what use did the governments make of such information laboriously collected and carefully tabulated. None! It did not need specially organised nutrition sections with trained nutrition officers to say that the diets of poor classes in India were defective or that their children were suffering from malnutrition. These facts virtually stared anyone in the face. The provincial nutrition organisations should have been used to plan and develop programmes to fight malnutrition. This was never seriously attempted. The governments provided funds just enough to permit the nutrition officers to tour round the province giving talks on nutrition, organising nutrition exhibitions and to publish pamphlets in local languages in addition to carrying out nutrition and diet surveys. The governments probably had a naive belief that that was all that was expected of them. There have been halfhearted attempts to provide mid-day meals to school children, distribution of vitamin tablets, food yeast, skim milk, etc., but not one state government has pursued with vigour and consistency any measures calculated to improve the nutrition of the people they governed.

It may be argued that the problem of improving nutrition of the Indian population is so vast that the ordinary resources of any government are not likely to meet the needs of the situation. Granted that it is so, there is another side of the picture which must not be ignored. There were and still are certain pressing problems of limited scope, crying for relief. Has any State done anything worth describing to solve them? We can give three examples, endemic goitre in the Himalayan and Sub-Himalayan regions, beri-beri in the Northern Circars and fluorosis in Nellore and nearby districts in Madras State. There is enough

scientific knowledge present to enable the formulation of plans for the control, if not the eradication of these diseases. There are obviously difficulties in the way, but these difficulties can only be overcome if work is taken in hand to solve the problem. Merely sitting back taking a count of difficulties and seeking shelter behind the ever present financial stringency are not going to make the problem any easier to solve. What is needed is careful thought, a full appreciation of the damage done by continuing a policy of *laissez faire* and a resolution to remove the blot of such preventable diseases as have been mentioned above. After all, it cannot be said that governmental efforts have eradicated either cholera, plague, or small-pox, but the governments are going on with the schemes for their control and eradication. Success has been only partial and slow to come by, but few people can accuse the government of neglect in the matter. Unfortunately, the same cannot be said where malnutrition and specific nutritional diseases are concerned. Under the present circumstances it is even difficult to visualise a time when the state will fully realise their responsibility and stir themselves to action. It is, therefore, all the more necessary to combine educating our legislators and executives so that they may know that their hopes of building a virile nation and of improving standards of life will bear no fruit unless the health of the people and their correct nutrition are ensured for all time.

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## CHAPTER XXIII

### POPULATION AND FOOD SUPPLIES

NO DISCUSSION on nutrition in India would be complete without due consideration being given to the pressing question of our increasing population in relation to available and potential food supplies. Fears have been expressed both within the country and without that even today the country is incapable of supporting its present population. The population pressure is undeniably increasing and is bound to affect adversely any prospect of improving the nutrition of Indian people. The questions which face us today are: (i) is the present population excessive in relation to land resources, (ii) is the rate of increase alarmingly great and (iii) would the progressive shrinking of land area per capita militate against the solution of food problem of the Indian people? A full discussion of all these questions is not possible within the scope of this book. We do not claim intimate knowledge of the subject and hence the following discussion may appear to treat the problem only superficially. We only intend to state certain facts and will attempt to draw certain conclusions arising from them. For greater details the reader will have to refer to other books and treatises on the subjects.

#### POPULATION

There is little accurate information about the total population of the Indian sub-continent before the year 1872. According to certain rough estimates the population of India was approximately 100 millions at the beginning of the 17th century. It was only in



the year 1872 that the first organised census was taken, and after that it has been repeated in the first year of every new decade. Table No. XXX shows the increase in numbers and the rate of increase from 1872 to 1941.

TABLE XXX

## POPULATION OF INDIA IN RECENT YEARS

Census	Population millions	Increase per cent since previous census
1872	203	—
1881	250	23·2
1891	279	11·6
1901	284	1·8
1911	306	7·7
<u>1921</u>	<u>308</u>	0·7
1931	338	9·7
<u>1941</u>	<u>389</u>	15·1

On considering the records for the last seventy years (counting back from 1941) one finds that it was during the years 1921-1941 that the rate of increase was comparatively high. In the preceding three decades the population growth was slow on account of famines, epidemics of plague and cholera, and the pandemic of influenza which swept the world in 1918 and 1919. On the other hand, no major calamity struck India between 1921 and 1941 to impede the unchecked growth of population. It must be pointed out, however, that even during the years of accelerated growth, the annual rate of increase was about

1.5 per cent which in itself is not astonishingly high. It is the total number that is added every year, i.e., 3 to 4 millions, that makes the problem difficult. It is expected that for some time to come this tendency will continue. Evidence for this can be found in the birth and death rates as given in Table XXXI.

TABLE XXXI  
BIRTH AND DEATH RATES IN BRITISH INDIA

Period	Birth Rate per mille	Death Rate per mille
1891-1900	33	31
1901-1910	38	34
1911-1920	37	34
1921-1930	35	26
1931-1940	34	23
1941	32	22
1942	29	21
1943	26	23
1944	25	24
1945	27	25
1946	28	18

The figures given above are admittedly incomplete as they cover only British India and not the princely states. But when viewed over a period of fifty years or more they indicate the trend which could be considered to apply roughly to the rest of India as well. Death rate has shown a substantial decrease mainly owing to better health services and control of epidemics. Although the birth rate has

also shown a tendency to decrease, the latter is not so marked as that shown by the figures for death rate. The age and sex composition of the Indian population is such that it is favourable for continued growth of the population. It is, therefore, certain that with continued improvement in health services the death rate would fall still further with the result that substantial increase in population is to be expected within the next generation at least unless any major calamity such as widespread famine or outbreak of any disease of pandemic dimensions intervenes. If the same rate of increase that held between 1922-1941 continues, it is estimated that the total population of the Indian sub-continent in 1951 will be about 447 millions. The partition of the country in August 1947 has made the problem more acute for India than for Pakistan, for nearly four-fifths of the total population was left within the borders of the Indian Union. The estimated population of the Indian Republic in 1951 will be in the neighbourhood of 356 millions. It is stated that with the improvement in the standards of living consequent on developing the industrial potential of this country, the birth rate would decrease, which in its turn will lower the rate of population increase. Even if this assumption is correct, the effect of industrial development on birth rate will require at least a generation, if not more, to make itself felt. This leaves us with the probability (for sometime to come, at least) of continuous growth of population at the rate which existed in the decades 1922-1941.

#### LAND IN RELATION TO POPULATION

The total land area of the Indian sub-continent was estimated to be about 1,011 million acres (1.58

million square miles). After the partition, the total land area of the Republic is estimated to be 781.4 million acres. The situation in relation to the population as roughly estimated in 1950 can be seen from the following figures:

(a) Total land area	....	781.4 million acres.
(b) Land not available for cultivation, uncultivable waste and land under forests	....	364.0      „      „
(c) Cultivated land, current fallow and cultivable waste	....	417.4      „      „
(d) Estimated population in 1950	....	351.0 million.
(e) Total land area per capita	....	2.2 acres.
(f) Land available for cultivation per capita	....	1.19      „
(g) Cultivated land per capita	....	0.78      „

Thus the total area of land that can be put under cultivation is limited to 1.19 acres per capita. There is a wide gap between the land actually cultivated and that which can be cultivated as a result of an all out programme. With the expected increase in population at the rate of approximately 5 millions per year, the total land area per capita is bound to shrink progressively and it is not certain where and when the relation between land and population will be stabilized.

The Report of the Famine Enquiry Commission (1944) refers to an estimate of an American agricultural

expert according to which the land needed per capita for various grades of dietaries is as follows:

Diet	Acres per capita
1. Emergency restricted diet ....	1.2
2. Adequate diet at minimum cost ....	1.8
3. Adequate diet at moderate cost ....	2.3
4. Liberal diet ....	3.1

There is no need to go into details about the composition of diets included in each category. It must be clear, however, that in India the actual cultivated land per capita is lower than that required to give the lowest quality diet, and the plan of putting under the plough the whole of cultivable land will, if successful, yield in India only 1.19 acres per capita. It is physically impossible to attain the figure of 2.3 acres/capita, for the total land area in India is 2.2 acres/capita with immediate prospect of shrinkage. In the absence of more detailed information about the American figures of land requirement it will not be wise to comment on them. Even assuming that they correctly represent the approximate requirements, the calculations must have been based upon the agricultural practices and productive capacity of the land in the U.S.A. Land in India has been under cultivation for centuries, it has been considerably impoverished owing to archaic practices and the yields have shown a tendency to decrease even over the last 50 years. Taking all these things into consideration one may probably be justified in assuming that the corresponding land requirements in India may have to be placed on a higher level, which will make the problem of adjusting land requirements to Indian population



well nigh impossible. There is one consideration, however, which may throw doubts on the applicability of the American figures of land requirements to India. The types of diets accepted as nutritionally adequate differ in U.S.A. and in India. In the former, greater emphasis is laid on animal foods such as milk, meat and eggs than on plant foods, whereas in India, the nutritionally adequate diets as recommended by the Nutrition Advisory Committee still lean heavily on cereals and other plant foods. While we do not dispute that the American type of adequate diet would be the goal worthy to strive for, conditions in India would not allow the attainment of that objective within any reasonable time. Our attempts must, therefore, be directed to suggest improvements within the existing framework of our dietary practices. Viewed from this standpoint with a greater emphasis on plant foods, it must be clear that our land requirements would probably have to be calculated on a different basis.

The problem of utilising the whole of land available for cultivation is taxing to the utmost the resources of various state governments and the Indian Central Government. Its successful solution depends upon India being able to find sufficient money to invest for the purposes of land development. There are other difficulties too which can only be barely recounted. Poverty, illiteracy and ignorance born of both exist in Indian masses on a colossal scale. Unscientific and outdated practices are extremely common in Indian agriculture. The fragmentation of land has resulted in small holdings which are unsuitable for large-scale farming. Vagaries of monsoon result in floods and droughts both of which are equally destructive. Soil erosion

proceeds unchecked. Deforestation has progressed without any attempts to control it till recently. These facts should give a rough measure of the immensity of the problem to be tackled.

### AGRICULTURAL PRODUCTION

The estimated production of foodstuffs in India falls considerably short of our requirements. An estimate made in 1944 for undivided India is given in Table XXXII.

TABLE XXXII—ESTIMATED DEFICITS IN  
FOOD PRODUCTION

Article		Production Million tons	Deficit %
Cereals	...	60.0	10
Pulses	...	7.5	20
Fats and oils	...	1.9	250
Fruits	...	6.0	50
Vegetables	...	9.0	100
Milk	...	23.0	300
Meat, Fish and Eggs	...	1.5	300

The deficits in certain more important categories of foodstuffs are enormous ranging from 100 to 300 per cent. It must be mentioned that the partition of India has, if anything, made the situation worse by increasing deficits in almost every category of foodstuff. Herculean efforts are needed to make up the deficiency in order that Indian citizens may be assured of a nutritious diet.

The Government of India became suddenly and acutely conscious of the food situation in 1942-43.

The repercussions of World War II and the experience of the Bengal famine prompted the government to take action directed to the improvement of food supplies. As a first step the Government initiated the "grow-more-food campaign" throughout the country. The first object was to bring more land under cultivation and the second to take other necessary steps designed to increase food production, viz., digging wells, bunding of tanks for irrigation purposes, composting for manure, supplying of improved seed, etc. Under this scheme the area under cash crops such as cotton, jute, tobacco, etc., was not only controlled but some of it was diverted to food production. The campaign was apparently pushed with vigour and with a great deal of publicity and propaganda. The results of the campaign as assessed at the end of six years were extremely disappointing and in certain instances even calamitous. At the Ninth Conference of Indian Society of Agricultural Economics held in Hyderabad in December 1948 an unrelieved tale of woe testified to the fact that the campaign had been an unqualified failure. The experts dilated a great deal upon the causes of failure, but that was of no help.

In 1944, the Indian (Imperial as it then was) Council of Agricultural Research published a memorandum entitled "Agricological Development in India" in which detailed plans were described for post-war development of agriculture and animal husbandry. The non-recurring cost of the plan was estimated to be Rs. 1,000 crores (for British India alone). The Council had also prepared a skeleton plan designed to step up production by 100 per cent within 15 years after the end of the war, for which a recurring expenditure of 20 crores was estimated. This plan envisaged an era of peace and availability

of funds, both of which were denied to India due to developments which are too well known to require recapitulation.

The agricultural experts in this country thus did feel that India could be self-sufficient with regard to food. The Famine Enquiry Commission (1944) appointed to investigate the causes of food shortage in India and Famine in Bengal and to make recommendations as to the prevention of their recurrence, have also taken an optimistic view. They were of the opinion that India had the capacity to produce food not only sufficient to meet the needs of the growing population, but "enough to effect an improvement in the diets of the people". In the face of the opinion expressed by these two expert bodies we refrain from further comment. It is nevertheless true that judging from the present food supply situation the immediate prospect is none too bright.

In the above discussion will be found the answers for the two of the three questions posed in the opening paragraph of this chapter. It is comparatively less easy to find an answer for the third. It is, however, clear that as we are situated, the problem of improving nutrition in India appears considerably difficult. But we need not be despondent on that account. There may be ways of augmenting our food supplies. Firstly, there is considerable scope of increasing the yields per acre of various food crops. It is hoped that with the new irrigation, flood control and multipurpose projects the dependence of our agriculture on monsoons will considerably diminish. Projects are already on hand (a) to conserve waste for conversion to manure, (b) to distribute improved seed (c) to control plant diseases and pests, etc., all of which are calculated to increase the total food

produced in this country. It may also be possible to supplement locally grown foodstuffs by imports from countries which have excess to dispose of. This latter is bound up with the economic development of India and its increased capacity to purchase food from abroad. In short, the solution of the food problem of India depends upon a general all round economic and industrial development of the country. As such the subject assumes a very wide scope and it is time that we left it to experts to describe how it could be done.

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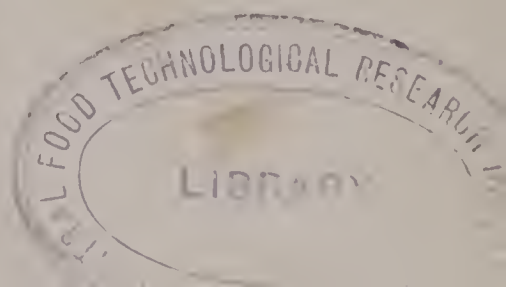
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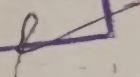


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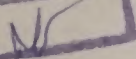
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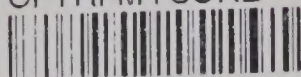
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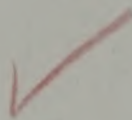
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